

STANDARDS DEVELOPMENT BRANCH OMCE



36936000005255

CATEGORIZATION (LISTING) BACKGROUND DOCUMENT - WOOD PRESERVING WASTES

MAY 1994

TP
996
.W6
C38
1994
MOE



**Ministry of
Environment
and Energy**

Copyright Provisions and Restrictions on Copying:

This Ontario Ministry of the Environment work is protected by Crown copyright (unless otherwise indicated), which is held by the Queen's Printer for Ontario. It may be reproduced for non-commercial purposes if credit is given and Crown copyright is acknowledged.

It may not be reproduced, in all or in part, for any commercial purpose except under a licence from the Queen's Printer for Ontario.

For information on reproducing Government of Ontario works, please contact Service Ontario Publications at copyright@ontario.ca

TP
996
.W6
C38
1994

Categorization (listing)
background document : wood
preservation wastes.
77264

ISBN 0-7778-2758-1

CATEGORIZATION (LISTING) BACKGROUND DOCUMENT

WOOD PRESERVING WASTES

SCI & TECH LIBRARY

MAY 1994



Cette publication technique
n'est disponible qu'en anglais.

Copyright: Queen's Printer for Ontario, 1994
This publication may be reproduced for non-commercial purposes
with appropriate attribution.

PIBS 3020

JP
996
W6
C2S
1994

TABLE OF CONTENTS

	PAGE
1.0 SUMMARY OF BASIS FOR CATEGORIZATION	1
1.1 Listing under Regulation 347	1
1.2 Listing under other Canadian legislation	2
1.3 Listing under U.S. legislation	2
2.0 DESCRIPTION OF INDUSTRY	2
2.1 Wood preservatives	3
2.1.1 Pentachlorophenol	3
2.1.2 Inorganic preservatives	3
2.1.3 Creosote	3
2.2 Wood preserving processes	4
2.2.1 Wood conditioning	4
2.2.2 Non-pressure treatment	5
2.2.3 Pressure treatment	5
2.3 Wood preserving industry in Ontario	6
3.0 WASTE MANAGEMENT PRACTICES	6
3.1 Waste generation	6
3.1.1 Wastewaters	7
3.1.2 Process residuals	8
3.1.3 Drillage and drillage residuals	10
3.2 Waste management	10
3.2.1 Wastewaters	10
3.2.2 Process residuals	12
3.2.3 Drillage and drillage residuals	12

4.0 HAZARDOUS PROPERTIES OF THE WASTE	13
4.1 Rationale for listing of wastes	13
4.1.1 Pentachlorophenol	14
4.1.2 Inorganic preservatives	15
4.1.3 Creosote	15
4.2 Hazardous characteristics	15
4.3 Physical and chemical properties	16
4.4 Environmental behaviour	16
4.5 Toxicity	16
5.0 CONCLUSIONS AND RECOMMENDATIONS	16
6.0 REFERENCES	18

APPENDICES:

A: Environmental behaviour and toxicity of pentachlorophenol

B: Environmental behaviour and toxicity of benzo(a)pyrene

C: Environmental behaviour and toxicity of 2,3,7,8-TCDD

D: Environmental behaviour and toxicity of arsenic

E: Environmental behaviour and toxicity of chromium

LIST OF TABLES

TABLE	PAGE
1. Wood preserving plants in Ontario	20
2. Estimated volume of treated wood in Ontario (1990)	21
3. Waste generation factors in wood preserving industry	22
4. Estimated volumes of waste generated by the Ontario wood preserving industry	22
5. Chemical analysis of soil samples	23
6. Wastes from wood preservation using pentachlorophenol	24
7. Inorganic wastes	25
8. Creosote wastes	25
9. Summary of vector scoring	26

1.0 SUMMARY OF BASIS FOR CATEGORIZATION

1.1 Listing under Regulation 347

The Ontario Ministry of Environment and Energy (MOEE) is considering amending Regulation 347 and listing the following waste streams from the wood preserving industry;

- a) *Wastewaters*, process residuals*, preservative drippage*, and discarded spent formulations from wood preserving processes at facilities that currently use or have used chlorophenolic formulations.*
- b) *Wastewaters*, process residuals*, preservative drippage*, and discarded spent formulations from wood preserving processes at facilities that currently use creosote formulations or formulations containing arsenic or chromium.*

* A detailed description of the waste streams mentioned above is presented in section 3.1 of this document.

The objective of this listing is to ensure that appropriate waste management of these waste streams is maintained within the Ontario industry and that new operations planning to establish in Ontario will manage these chemicals, including their waste streams, properly. Contamination of soil and groundwater has been identified from Ontario sites historically used for wood preservation. There is a potential for similar contamination in present and future sites, if these wastes are not managed properly.

Presently, one waste stream from the wood preserving industry is listed as Hazardous Industrial Waste in paragraph NA9316 of Schedule 1 (Regulation 347):

"Bottom sediment sludge from the treatment of wastewater from wood preserving processes that use creosote and/or pentachlorophenol."

Wastes from surface protection operations at sawmill sites are not included in this listing. The surface protection process is most commonly used at or near sawmill operations, and involves the application by spraying or dipping of sapstain control agents to freshly cut lumber. The purpose of surface protection is to prevent sapstain formation during short-term storage. The listing of waste streams from this process is deferred until sufficient information allows proper characterization of these waste streams.

1.2 Listing under other Canadian legislation

Under the federal Transportation of Dangerous Goods Act (TDGA), certain wastes from the wood preservative industry are classified as poisonous substances (Class 6.1, liquid or solid substances with a LD₅₀ (oral toxicity) of less than 50 mg/kg). Regulations under this Act require specific documentation (manifests and waybills), packaging and labelling for 9 classes of dangerous goods and wastes.

1.3 Listing under U.S. legislation

The United States Environmental Protection Agency (USEPA) has listed, under Section 3004, Part 261.32 of the Resources Conservation and Recovery Act (RCRA), bottom sediment sludge from the treatment of wastewaters from wood preserving processes that use creosote and/or pentachlorophenol. That listing was made effective pursuant to an interim final regulation promulgated in 1980 (U.S. Federal Register, 1980). In a new listing (U.S. Federal Register, 1990), the USEPA listed, as hazardous wastes, wastewaters, process residuals, preservative drippage, and discarded formulations from wood preserving processes generated at plants that:

- a) currently use, or have used, chlorophenolic formulations;
- b) presently use creosote formulations;
- c) presently use inorganic preservatives containing arsenic or chromium.

2.0 DESCRIPTION OF INDUSTRY

Bio-deterioration of wood occurs through attack by fungi, bacteria, insects and minor micro-organisms. In a temperate climate, fungi are the most important cause of wood deterioration. There are four major requirements for fungal growth:

- a) a food source, i.e. cellulose, lignin or other wood constituent;
- b) a substrate moisture content of between 25 and 100%;
- c) a temperature between 10 and 35°C;
- d) an adequate supply of oxygen.

Fungicides are used in the treatment of wood in order to preserve the wood from the attack of fungi. The fungicides most commonly used in Ontario are pentachlorophenol, inorganic preservatives (such as chromated copper arsenate), and creosote (see Section 2.1 for details).

2.1 Wood preservatives

2.1.1 Pentachlorophenol

Pentachlorophenol is one chemical from a group of synthetic organic compounds, called chlorophenols. Chlorophenols are commercially manufactured by reacting chlorine with phenol. Pentachlorophenol formulations, ready to be applied to wood, typically contain 5 percent total pentachlorophenol. However concentrations may range from 2 to 9 percent. Pentachlorophenol solution has been found to be contaminated with all congeners of polychlorinated dibenzo-p-dioxins (PCDD) and dibenzofurans (PCDF) (USEPA, 1987; EPS, 1988b).

2.1.2 Inorganic preservatives

The elements of concern in the inorganic preservatives consist of arsenical and chromate salts dissolved in water. The inorganic salts most commonly used in the wood preservation are chromated copper arsenate (CCA, mixture of CrO_3 , CuO and As_2O_5), ammoniacal copper arsenate (ACA), acid copper chromate (ACC), chromated zinc chloride (CZC), and fluor-chrome-arsenate-phenol (FCAP). During the treatment process, CCA stock solution is typically diluted to 1 or 2 percent total CCA concentration.

Production of wood treated with inorganic preservatives has more than doubled since 1980. This trend is due to the increased use of treated lumber for decks, porches, and other exterior, weather-exposed structures. As a result of this increase, particularly for CCA use, most new facilities are CCA plants. Facilities that use exclusively inorganic preservatives are (on average) only 8 years old.

2.1.3 Creosote

Creosote generally refers to mixtures of relatively heavy residual oils (liquid and solid aromatic hydrocarbons) obtained from the distillation of wood, coal tar, or crude petroleum. Only creosote from coal tars are used as wood preservatives in Ontario (CSA, 1989).

2.2 Wood preserving processes

All wood preservatives are applied to wood using similar processes. The wood is normally pre-treated, or conditioned, in order to improve the penetration of the chemicals in the wood. A limited quantity of wood is preserved using non-pressure treatment processes in which the preservative is allowed to diffuse into the wood. Pressure treatment takes place in sealed pressure vessels known as cylinders or retorts.

2.2.1 Wood conditioning

The chemical penetration required to preserve the wood adequately can only be achieved if the wood has been properly conditioned: the moisture content of the freshly cut wood must be reduced to a point where the preservative can penetrate the wood and be retained there. Conditioning is required before pressure, or even non-pressure preservation processes are applied.

Conditioning or moisture reduction methods include:

- a) air seasoning (wood drying under natural atmospheric ventilation);
- b) kiln drying (wood heated in a kiln);
- c) steam drying (steaming the wood at elevated pressure in a retort followed by application of a vacuum);
- d) Boulton process (heating the wood under reduced pressure in a retort);
- e) vapour drying (heating the unseasoned wood in a solvent under pressure).

Air seasoning and kiln drying generate little or no amount of wastewater, because of water evaporation. Facilities that use these conditioning methods, principally non-pressure treaters and facilities that use inorganic preservatives, generate little wastewater.

Steam drying, Boultonizing and steam conditioning produce wastewater mostly consisting of water driven out of the wood. Steam conditioning also produces a considerable volume of steam condensate. The volume of water from wood conditioning varies according to wood type, preservative formulation, conditioning method, and other factors (USEPA, 1987).

After the moisture content of the wood has been reduced by conditioning, the wood is preserved either by using simple non-pressure methods or using pressure processes.

2.2.2 Non-pressure treatment

Non-pressure processes include brushing, spraying, dipping, soaking, and thermal treatment. These processes involve the repeated use of preservative in a treatment tank with fresh preservative solution added to replace consumption loss. The continual reuse of preservative leads to the accumulation of wood chips, sand, stones, and other debris contaminated with various hazardous constituents in the bottom of the treatment tanks. This is the major source of process waste for non-pressure processes.

In the full length thermal process, dried wood products are laid horizontally in open-top tanks and secured to prevent floating. Hot oil preservative, generally at temperatures of 93-113°C, is pumped into the tank until the wood is completely immersed in the solution. The solution is maintained at 93-113°C for a period of six hours or longer, after which the hot solution is pumped back to the storage tank. This hot solution heats up the air pockets in the wood, causing the wood to expand. Immediately, the tank is flooded with a similar cold solution (32-65°C) which causes a contraction of the hot air in the cells, creating a vacuum. The result is a further penetration of the solution into the wood. Thermal butt treatment is a similar treatment process for the ground line area of a pole (CSA, 1989).

2.2.3 Pressure treatment

There are two basic types of pressure treatment processes, based on the sequence in which vacuum and pressure are applied: empty-cell process and full-cell process (the terms "empty" or "full" refer to the level of preservative retained by the wood cells).

The first method, referred to as the "empty-cell" process, is used to obtain relatively deep penetration with limited absorption of preservative. Although there are numerous empty-cell processes, generally the preservative is pumped into the treating cylinder or retort at the same time as air pressure is applied. The air enters the cells of the wood. Once the desired level of retention has been achieved, the unused preservative is drained off and the excess preservative is vacuum pumped away from the wood. Any preservative that had entered the cells is driven out by the air within the cells, due to the release of pressure.

The second method, known as the "full-cell" process, results in higher retention of preservative but in lower penetration, when compared to the empty-cell process. A high vacuum is created in the treating cylinder in order to remove the air within the cells and to allow the preservative to penetrate in the wood. The preservative is pumped in the cylinder without breaking the vacuum. Once the cylinder is full, pressure is applied until the targeted level of preservative retention is achieved. Preservative adsorption is monitored during the treatment. There is no difference in the type of wastes generated by the two processes, although wood treated by the full-cell process may produce more drippage.

2.3 Wood preserving industry in Ontario

Table 1 summarizes the status of the wood preserving industry in Ontario. In an audit program, done in 1991 with the cooperation of Environment Canada (Brown, 1991), 20 known wood preservers were identified operating in Ontario. The volume of the retorts was estimated to the best knowledge of the individual plant operators. The retort volume is an indication of the capacity of production of a plant: the estimated retort volumes range from 24 to 719 cubic meters. The majority of the plants are less than 20 years old, with 40% of them built in the last 10 years (i.e. after 1980). The majority of the plants (i.e. 19 out of 20) use chromated copper arsenate (CCA) as preservative. Four (4) plants use pentachlorophenol, and 2 use creosote. Some plants use more than one type of preservative.

The production level (i.e. volume of wood treated) of a plant depends not only on the retort volume but also on the layout of the plant (efficiency in material handling and shift scheduling) and the market conditions. Some plants did not report their production level in the audit. Individual production (or treatment) factors (i.e. annual volume of wood treated per volume of retorts) were derived from reported values. The average production factor was then used to estimate the production levels of the other plants, using their individual retort volume. Estimates of the production of treated wood are presented in Table 2. An estimated total of near 1.6 million cubic meters of wood has been treated in Ontario in 1990. The estimate is based on data from the Environment Canada audit (Brown, 1991) for CCA plants, and on a report of the wood preserving industry in the United States (Micklewright, 1987) for creosote/PCP plants.

3.0 WASTE MANAGEMENT PRACTICES

3.1 Waste generation

Wastes generated from wood preserving processes consist of wastewaters, process residuals (including discarded preservatives) and drippage.

The listing includes waste streams from facilities that previously used chlorophenolic formulations because of potential contamination by dioxins and/or furans. The source of contamination may be from the plant or from the equipment itself. In order to exempt these waste streams from the first listing paragraph (chlorophenolic formulations), proper cleaning of the site and/or replacement of equipment must be done according to procedures suggested in U.S. Federal Register (1990) and acceptable to MOEE. However, the waste streams remain listed under the second listing paragraph, if the facilities are using creosote formulations or formulations containing arsenic or chromium in their wood preserving operations.

The waste generation factors, used for estimating the amount of waste generated in various waste streams, are presented in Table 3. They are derived from information collected during the 1991 Ontario audit (Brown, 1991) and from results of a survey conducted for the U.S. wood preserving industry (USEPA, 1987). Table 4 gives the estimates of the annual waste generation from the wood preserving industry in Ontario. An estimated 21,314 kl (kl = 1000 liters) of wastewater, 328 kl of wastewater treatment residuals, 206 kl of process residuals and 271.9 kl of drippage were generated in 1990.

3.1.1 Wastewaters

Wastewaters subject to this categorization (listing) include:

- a) residual water generated during steam conditioning of wood (see Section 2.2.1) in treatment cylinders, prior to applying preservative (this water is generally contaminated with preservative residuals in the cylinders);
- b) residual water from preservative recovery and regeneration;
- c) water used to wash excess preservative from the surface of preserved wood (especially poles);
- d) condensate from kilns used to dry preserved wood;
- e) wastewater from operations involving the rinsing of drums, storage tanks, equipment and process area;
- f) water and rainwater that accumulate in door seals and retort sumps, and rainwater falling on, or in the immediate vicinity of, the treating cylinder and work tank area.

Wastewaters that do not come in contact with chlorophenolic formulations, creosote formulations, inorganic formulations of arsenic and chromium, or listed wastes from wood preserving plants are not included in this categorization (listing).

Examples of wastewater not included in this listing are:

- a) condensate from boilers, which do not come in contact with process contaminants;
- b) condensate from drying kilns used to dry untreated wood. The kilns must have never been used to dry treated wood;
- c) wastewater generated from steam conditioning untreated wood in cylinders. The cylinders must have never been used to steam condition treated wood;

- d) rainwater that is collected in a fashion that keeps it segregated from preservative formulations or listed wastes from wood preserving plants.

The above streams, if collected and treated with process water, are subject to the "mixture rule" of Section 1 (ss 27) in Regulation 347. For example, rainwater collected on drip pads and conveyed to associated collection systems would be considered a hazardous waste because it comes in contact with listed wastes (such as drippage, process residuals, and wastewaters) from wood preserving operations.

Wastewater and wastewater discharge from wood preserving processes are currently controlled under the Ontario Water Resources Act (OWRA) through the issuing of certificates of approval. The proposed listing will also regulate wastewater from wood preserving under Regulation 347 but not wastewater discharge.

As quoted from Section 16 (1) (b) of Regulation 347 "No generator shall permit liquid industrial or hazardous waste to pass from his control or leave the waste generation facility except, by direct discharge to a sewage works subject to the OWRA." Therefore wastewater discharge from an OWRA approved wastewater treatment system in the wood preserving industry is exempt under Regulation 347. The exemption would not extend to activities that occur prior to wastewater treatment. Hence tanks and other units in which wastewater is collected, stored, accumulated, and/or disposed at a wood preserving facility would be subject to Regulation 347.

3.1.2 Process residuals

Materials such as sawdust, wood chips, sand, dirt, and stones can be attached to the wood when it enters the retort (in the case of pressurized preservation processes), or the dip tank (in the case of non-pressurized processes). These materials may be washed off the wood during the process and may form a residue in the retort or in the dip tank. They may also settle out of the preservative solution elsewhere in the process (e.g. in work tanks or sumps) or be removed during filtration of the preservative prior to its reuse. Tar and emulsified or polymerized oils may also settle out in the treating cylinder, treating tank or dip tank, during wood preserving operations using creosote or pentachlorophenol. All of these wastes are considered process residuals.

Wood preservers typically recycle preservatives from various tanks (storing, mixing and working tanks). Preservatives lost with wastewater or through drippage into the sumps are also sometimes collected and reused in the process. The continual reuse of preservatives leads to accumulation of residues (such as sawdust, wood chips, sand, dirt, stones, tar, and polymerized oils) in the tanks. This material comprises the bulk of wood preserving process residuals (not including wastewater) and is collected in treating cylinders and tanks.

Specifically, process residuals subject to categorization (listing) include:

- a) precipitates from preservative solutions, tar, emulsified poly-merized oils;
- b) spent or discarded formulation;
- c) sediments from treating cylinders, treating tanks, and dip tanks;
- d) residuals, such as soil, wood extract, sawdust, wood chips, storage tank and cleanup sludges
 - from filter, screening, or exhaust control from spray booths;
 - from kilns used in the drying process of preserved wood;
 - from tanks used in holding, storing, working, mixing or general handling of preservatives;
 - accumulating in secondary containment surrounding tanks;
 - from door or cylinder sump pumps;
 - from recycling and regeneration of preservative;
 - from maintenance and cleaning of process equipment;
 - from hand spraying operations.

A review of the Generator Registration Reports in 1991 (GRR, 1991) indicates that 77,732 kg/yr of hazardous wastes (estimated by the generators as average annual quantity) are registered by the wood preserving industry. A total of 7,315 kg/yr are registered as being generated from out-of-province. These hazardous wastes are described by the generators as "soil, wood extract, sawdust, wood chips, storage tank and cleanup sludges". Under this proposed listing, these residuals would be considered listed (Schedule 1) hazardous wastes.

The total amount of registered waste generated in Ontario which would be considered process residuals is 70,417 kg/yr (GRR, 1991). The estimate of the total process residuals produced in the Ontario wood preservative industry (see Table 4) is 206 kL/yr. Assuming an average bulk density of 640 kg/kL (USEPA, 1987), it is estimated that 131,184 kg/yr of process residuals are generated. This estimate is 86% above the value reported by the generators in their GRRs. The actual volume of process residuals most likely resides between these two numbers, due to the fact that all of these wastes are not recognized as hazardous by all generators.

3.1.3 Dripage and dripage residuals

After treatment, some unabsorbed preservative formulation adheres to the treated wood surface. Eventually, this liquid drips from the wood or is washed off by precipitation. Immediately after the wood has been pressure treated, excess preservative will also exude from the wood as it gradually returns to atmospheric pressure (a phenomenon commonly called kick-back). Preservative formulations (specially pentachlorophenol and creosote) may continue to exude or bleed from treated wood for long periods, even after the wood is shipped off-site and installed for its intended end use. Dripage may be minimized by steaming the wood after treatment to wash off adhering preservative or by applying a final vacuum to the wood charge before it is removed from the retort.

Dripage and dripage residuals may accumulate on designated drip areas, on pathways over which treated wood is transported and in treated wood storage yards. Dripage and dripage residuals include:

- a) free dripage of preservative from treated wood;
- b) preservative that is washed off treated wood by precipitation;
- c) residuals from collecting and recycling preservative that drips off or is washed off treated wood.

Treated wood is stored in large storage yards, after the wood has been allowed to drip sufficiently over drip pads. Sufficient dripping is defined in EPS (1988a, b, c) recommendations for operation. If the recommended procedures are followed for the dripping process over the drip pads, minimal amount of dripage should occur in the storage yards.

Although dripage from the treated wood is considered hazardous waste, the practical approach dictates that *de minimis* concentrations of preservative cannot be controlled further in the storage yards, or in the applications or uses of the treated wood. The operator of the wood treating plant must however have a contingency plan for controlling excessive dripage, incidental dripage and spills in the storage yard.

3.2 Waste Management

3.2.1 Wastewaters

In 1991, three plants were discharging treated wastewater from their operation. All other plants did not dispose of their wastewater because it was stored or totally re-used in the solution make-up tank. The wastewater treatment systems utilized by the Ontario plants can be illustrated in the 5 following typical plants:

Plant A

The wastewater treatment system consists of one 44 ft³ oil/ water separator, followed by two (6'x 12' long) tanks connected in series. The first tank acts as a secondary gravity separator. The underflow goes to the second tank where steam lines evaporate the water into the atmosphere. The sludge is disposed of as a Schedule 1 waste (bottom sludge from wastewater treatment in PCP/creosote wood treatment). Under this proposed listing the wastewater fed to the evaporator tank would be also considered as a Schedule 1 waste.

Plant B

The wastewater is treated initially by two gravity oil/water separators. The oil is recycled to the treatment process. The treated wastewater is transferred to a flow equalization tank for pH adjustment and nutrient addition. The wastewater is further treated in a biological activated sludge treatment system, consisting of an aeration tank and a clarifier. The sludge from the clarifier is recycled to the aeration tank and the water is discharged as industrial effluent, meeting the limits of the Ontario Water Resources Act. Surface runoffs are also collected and treated.

Plant C

This plant has two wastewater treatment systems: one for surface runoffs and a second one for process wastewater.

Surface runoffs are collected by a retaining wall around the process area. The suspended solids are removed by a DAF (Dissolved Air Flotation) unit. Two carbon columns further remove the hazardous contaminants. The slurry coming from the DAF unit is dewatered on a drum filter (this filter sludge is considered a Schedule 1 waste). The wastewater from the carbon columns is discharged in the effluent.

The process wastewater is initially treated with a flocculating agent to remove suspended solids. Hazardous contaminants are removed from the treated wastewater by using an ultraviolet enhanced oxidation system and activated carbon columns. The sludge from floc treatment is a Schedule 1 waste. The water from the carbon columns is discharged as an effluent.

Plant D

Cedar and lodgepine poles, which have been dried off site, are thermally treated with pentachlorophenol. Wastewater is generated from rainwater, which accumulates in the open working tanks. A new evaporator system (approved in 1991) treats this wastewater. Oil is recycled in the plant and recovered condensed water is re-used in the CCA circuit.

Since minimal amounts of wastewater are generated, the wastewater was stored on site. However a new evaporator system (approved in 1991) treats this wastewater. Oil is recycled in the plant and recovered. Condensed water is re-used in the CCA circuit.

All other plants

The wastewater generated in all CCA plants in Ontario is re-used in working tanks to dilute the preservative concentrate (typically 50%) to the working solution (typically 2%).

3.2.2 Process residuals

From the review of the Generator Registration Reports (GRR, 1991), the plants have registered the following process residuals as hazardous wastes:

- 1) Hay filter medium;
- 2) API separator sludge;
- 3) Activated carbon;
- 4) Sludge from non-pumpable tank and system clean up;
- 5) Sludge from PCP storage tank (5 % PCP).

Some of these wastes (1, 2 and 3) are considered to be sludges from a wastewater treatment system and therefore classified as a Schedule 1 waste. The last two wastes (4 and 5), although it could be argued that they are not generated technically from a wastewater treatment system, have also been classified as Schedule 1 wastes. With this proposed listing, waste 4 and 5 would be considered process residuals and would be classified as Schedule 1 waste from specific sources.

All the above process residuals were disposed of by certified carrier and receiver in 1991.

3.2.3 Dripping and dripping residuals

Management of dripping varies from facility to facility and often depends on the production schedule. A facility producing near its capacity generally removes the charge from the treating cylinder and quickly transfers it to the dripping area and/or shortly later to the storage yard. In this case, excessive dripping may occur in the treated wood storage yard. At facilities operating on slower schedules, a charge may be allowed to drip in the retort for an extended period of time. Other facilities have drip pads over which trams of freshly-treated

wood are allowed to sit for one to three days. Drillage may be routed to a sump and reused in the treatment process or may be discarded. Certain facilities have fixation plants where water is driven off, consequently decreasing the fixation and drillage time.

All CCA plants report using a drip pad, averaging 822 m² in area. The reported time allowed on the pad for fixation average 64 hours. The reported time ranges from a period of 5.5 hours to a maximum of 120 hours (Brown, 1991). Environment Canada (EPS, 1988c) recommends a fixation time of 48 hours under cover, and 96 hours for uncovered areas. Four plants have some type of fixation process which dries the treated wood, thus reducing the required fixation time. No plant has storage pads.

These is a serious potential for soil and groundwater contamination at plants without proper dripping period at the dripping pad before storage at the storage yard, or inadequate dripping pads. This potential for contamination has been confirmed at 3 different plants in the past.

Only one CCA plant reports that a spray wash is used as the treated wood leaves the retort. All CCA plants report that a final vacuum is used.

The practises used in the United States are summarized in the U.S. Federal Register (1988). In the U.S., 40 to 50 percent of the facilities that use pentachlorophenol and/or creosote have a paved drip pad. Near 91 percent of the facilities using inorganic preservatives have a paved drip pad. This larger fraction is believed to be due to the fact that their plants are generally newer than other wood preserving facilities and are specifically designed for recovery and reuse of preservative drillage. Drip pads are used to collect excess preservative that drips from the treated wood when it is removed from the treating cylinder.

Also in the United States, only 12 to 13 percent of the facilities treating with pentachlorophenol and/or creosote have storage pads (i.e. long-term storage yards) while 38 percent of facilities treating with inorganic preservatives have storage pads. The USEPA concluded that the remainder of the facilities (about 88 percent of pentachlorophenol and creosote facilities and 62 percent of the inorganic facilities) allows any preservative that drips off stored, treated wood (and any preservative that is washed off by rain or snow) to be disposed on the ground.

4.0 HAZARDOUS PROPERTIES OF THE WASTE

4.1 Rationale for listing of wastes

The rationale for listing the wastes under consideration is based on a number of contaminants of concern found in the wastes: pentachlorophenol, benzo(a)pyrene, 2,3,7,8-TCDD (equivalent toxicity), arsenic and chromium. The selection of the five contaminants is based on their level of toxicity and their relative concentration in the wastes. The behaviour of the contaminants in the waste streams is detailed in the appendices of this document.

Other contaminants may be present in these waste streams. Although their toxicity may be as important as the toxicity of the five selected contaminants, their concentrations are of less concern.

Table 5 summarizes the results of the chemical analysis of soil samples taken at various CCA plants during the Environment Canada audit (Brown, 1991). The audit covers 19 of the 20 plants identified in Ontario. Information about the size and type of operations, management of materials (wood, chemicals and wastes) are identified in the audit form. Soil samples were taken at the majority of the plants (from drip pad, storage and off-site areas with no obvious contamination). Process residuals, surface and ground water were also sampled at most plants. Contamination of soil samples collected near the operation is an indication of some level of mismanagement of the wastes.

A U.S. study, conducted in 1987 (Jenkins, 1987), gives some results for the composition of the waste streams. The study includes a survey of 100 plants (of the 709 plants identified) and sampling of waste streams in 9 of the 100 plants surveyed. Results of this study are presented in Tables 6, 7 and 8. Concentrations of specific contaminants in specific waste streams are reviewed in the following sections.

4.1.1 Pentachlorophenol

Waste generated from the preservation of wood using chlorophenolic formulations contain high concentrations of chlorinated phenols (or chlorophenols) and predominantly pentachlorophenol (PCP). Results of 3 waste streams using this process are presented in Table 6. PCP concentrations have been measured as high as 310 ppm in wastewaters, 3.4 % in process sludges or residuals, and 5.2 % in drippage.

Pentachlorophenol formulations are also contaminated with polychlorinated dibenzo-p-dioxins (PCDDs) and polychlorinated dibenzo-furans (PCDFs). Dioxins or furans have been measured up to 300 ppb in wastewaters, 140 ppm in process sludges and up to 100 ppm in drippage (see Table 6).

In 1992, the Ministry of Environment and Energy (MOEE, 1992) sampled soils at a pentachlorophenol plant. The results indicated that four soil samples in the storage area were found to be contaminated to a level of 80 to 450 ug/g pentachlorophenol and 2.72 to 14.32 ng/g (TEQ) dioxin/furan. This contamination was restricted to hexa, hepta and octa congeners. The CCME Interim Canadian Remediation Criteria for PCP in industrial soils is 5 ug/g. There is no industrial soil criterion for dioxin at present. However the residential soil criterion is set at 1 ng/g TEQ.

4.1.2 Inorganic preservatives

Waste from the preservation of wood with inorganic formulations contain high concentrations of copper, arsenic and chromium. Table 5 summarizes the results of soil testing at 17 plants in Ontario. Copper concentrations have been measured up to 2,000 ppm near the drip pad area, 193 ppm off site and 4220 ppm in storage area. Arsenic levels up to 2,430 ppm near drip pads and 63,632 ppm in storage area have been measured. Total chromium level up to 1,920 ppm near drip pad, and 5,600 ppm in storage area have also been measured.

Results of the U.S. survey (Jenkins, 1987) are presented in Table 7. Results of samples collected from monitoring wells and surface waters indicate the potential for ground water contamination. At one plant, a puddle in the storage yard contained high levels of copper (2,700 ug/L), arsenic (5,200 ug/L) and total chromium (68,200 ug/L). A monitoring well in another plant contained 134, 1,700 and 43 ug/L of the respective contaminants. A soil sample from a drip area of one plant had a copper, arsenic and total chromium concentration of 4,220, 63,632, and 5,600 ug/g respectively. The decommissioning guidelines are 225, 40, 750 ug/g respectively.

4.1.3 Creosote

Soil contamination in a pentachlorophenol/creosote plant has resulted in the surface water runoff requiring treatment. Results of analysis by Environment Canada in 1989 (Brown, 1990) of the untreated surface runoff averaged 6640 ug/L for chlorophenols, 630 ug/L for PAHs and 233 TEQ ng/L for dioxins/furans. Also untreated process wastewater was found to contain 110,106 ug/L chlorophenols, 29,489 ug/L PAHs, and 3,962 TEQ ng/L dioxins/furans.

Waste from the preservation of wood with creosote contain high concentrations of poly-aromatic hydrocarbons (PAHs). In a USEPA (1987) study, 16 PAHs and 6 phenolics were identified. Table 8 summarizes the concentration of the 8 selected constituents of concern from that treatment process. B(a)P was selected as the indicator for the PAHs, and a toxicity profile is presented in Appendix B.

4.2 Hazardous characteristics

The wastes generated in the wood preserving industry do not exhibit the following hazardous characteristics: corrosivity, reactivity, ignitability.

However, the wastes generated from the preservation of wood using inorganic formulation exhibit, at one point or another, the hazardous characteristic of leachate toxic for arsenic and total chromium.

As indicated in Tables 6, 7 and 8, there are several constituents of concern in the proposed waste streams. For this listing, specific chemicals have been chosen to represent the family of chemicals. PCP and B(a)P are representative contaminants found in PCP treatment. Various type of dioxins found in PCP wastes are expressed in terms of equivalent toxicity of 2,3,7,8-TCDD. Arsenic and chromium are the major constituents of inorganic preservatives. B(a)P is also a good indicator for creosote wastes, although benzo(a)anthracene is the major contaminant according to Table 8. This selection of 5 contaminants does not indicate that the other chemicals are not to be considered in a delisting application.

4.3 Physical and chemical properties

A selected number of physical and chemical properties of each of the contaminants of concern is presented in the individual Appendices.

4.4 Environmental behaviour

The environmental behaviour of the contaminants is expressed in terms of their mobility in different media, their persistence or half-life in the environment, and their potential to reach a receptor. Detailed analyses of the five contaminants of concern are presented in Appendices A to E, at the end of this document. The results of the analyses are summarized in Table 9.

4.5 Toxicity

The toxicity of the 5 contaminants of concern is reviewed in details in the individual appendices attached to this document. The main source of information for evaluating the toxicity of these compounds is derived from Preliminary Assessments (which include various literature searches) prepared by the Hazardous Contaminants Branch (MOEE) and toxicological profiles prepared by the Agency for Toxic Substance and Disease Registry, U.S. Department of Public Health and Human Services.

5.0 CONCLUSIONS AND RECOMMENDATIONS

It is estimated that near 1.6 million cubic meters of wood is treated with preservatives annually in Ontario. Some 20 Ontario plants produce annually an estimated 21.3 million liters of wastewater and near 0.8 million liters of residuals and drippage. These wastewater and residuals are contaminated with either pentachlorophenol, benzo(a)pyrene, arsenic, chromium or residual dioxins and furans.

Unless these wastes are re-used within the plant, they must be managed safely, because they contain sufficient concentrations of toxic contaminants which could adversely affect human health and the environment if allowed to enter the environment. For example, soil and groundwater contamination has been observed in some Ontario plants, because of mismanagement of either process residuals and drippage, or wastewaters.

Under this new listing, waste streams (i.e. wastewaters, process residuals, preservative drippage and discarded spent formulations) are identified in addition to the existing listing of bottom sediment sludge (NA9316). This new listing will inform generators in the industry regarding the hazardousness of these wastes, and will require management under Regulation 347. Listing of these new wastes streams will therefore reduce the potential for further contaminations at these sites and control the potential for contaminations at present and future uncontaminated sites.

These contaminants are generally very mobile and persistent in the environment. The most common route of environmental exposure is through contamination of soil, and possibly groundwater, surrounding the wood preserving plant. Soil contamination from this route has been documented in Ontario.

These 5 contaminants of concern are, in general, relatively very toxic in terms of acute lethality, sublethal effects on mammals, mutagenicity and carcinogenicity.

These considerations, mentioned above, justify that wastewaters, process residuals, preservative drippage and discarded spent formulations from the wood preserving industry be listed in Schedule 1 of Regulation 347.

6.0 REFERENCES

Bérard, Marie-France and Thomas Tseng, 1986.

Survey of Northern Wood Preservers Inc., Thunder Bay, Ontario, October, 1984 (unpublished report). Environment Canada, 25 St. Clair Ave. East, 7th Floor, Toronto, Ontario, M4T 1M2.

Brown, P, 1990.

Personal communication with Pauline Brown, Environment Canada, Toronto.

Brown, P, 1991.

Personal communication with Pauline Brown, Environment Canada, Toronto.

CSA, 1989.

Canadian Standards Association, Wood Preservation Commodity Standard CAN/CSA-080.10-M89.

EPS, 1988a.

Creosote wood preservation facilities: recommendations for design and operation. Prepared by Envirochem Services, for the Wood Preservation Industry Technical Steering Committee, Ottawa, 1988, 90p, EPS 2/WP/1.

EPS, 1988b.

Pentachlorophenol (PCP) wood preservation facilities: recommendations for design and operation. Prepared by Envirochem Services, for the Wood Preservation Industry Technical Steering Committee, Ottawa, 1988, 90p, EPS 2/WP/2.

EPS, 1988c.

Chromated copper arsenate (CCA) wood preservation facilities. Prepared by Envirochem Services, for the Wood Preservation Industry Technical Steering Committee, Ottawa, 1988, 81p, EPS 2/WP/3.

GRR, 1991.

Generator Registration Reports (under Regulation 347) are kept in files at the Waste Management Branch, Ontario Ministry of Environment and Energy, 2 St. Clair Ave. W., 14th Floor, Toronto, Ontario, M4V 1L5.

Jenkins, C., 1987.

Background document supporting the proposed listing of wastes from wood preservation and surface protection processes. Final engineering analysis: Volume 1, Volume 2 (Appendices). Prepared by Radian Corporation (Virginia), for Dr. Cate Jenkins, USEPA, Office of Solid Wastes, Washington, D.C., July 1987.

Sheild, J.K., 1976.

Control of preservative wastes from wood treatment. Eastern Forest Products Laboratory Report OPX163E, 26 pages.

Statistics Canada, 1986.

Wood industries, Annual Census of Manufactures, Catalogue 35-250, ISSN 0835-0078.

Micklewright, J.T., 1987.

Wood Statistics 1985. A Report to the Wood Preserving Industry in the United States. American Wood Preserver's Institute, January, 1987.

MOEE, 1992.

Technical memoranda from W.I. Gizyn to R.G. Pearson, Phytotoxicology Section, Air Resources Branch, MOEE, May-April 1992.

USEPA, 1987.

Background document supporting the proposed listing of wastes from wood preservation and surface protection processes. Part one: final engineering analysis. Volume 1 and Volume 2 (appendices). Prepared by Radian Corporation, McLean, Virginia, for Office of Solid Waste, USEPA, Washington. July, 1987.

U.S. Federal Register, 1980.

Vol. 45, p. 33084.

U.S. Federal Register, 1988.

Proposed Rule. Vol. 53, No. 251 (Friday, December 30, 1988), p. 53282-53330.

U.S. Federal Register, 1990.

Final Rule. Vol. 55, No. 235 (Thursday, December 6, 1990), p. 50450-50490.

Table 1: Wood preserving plants in Ontario

	Plant	Location	Volume of Retorts (m ³)	Start Up Date	Process (a)
1	Brandon Forest Products Ltd.	Scarborough	68.85	1984	CCA
2	Coventry Forest Products Inc.	Bolton	80.06	1987	CCA
3	Domtar Inc.- Domtar Chemicals Group (Wood Preserving Division)	Trenton	292.05	1913	PCP/Creosote
4	Fryer Forest Products	Monetville	104.08	1984	CCA
5	Great West Timber Ltd.	Thunder Bay	107.28	1975	CCA
6	Guelph Utility Pole Company Ltd.	Guelph	213.85 (b)	1974	PCP/CCA
7	Hilan Wood Preservers	Kemptville	64.05	1977	CCA
8	Jan Woodlands Ltd.	L'Amable	104.08	1961	CCA
9	John A. Biewer (Canada) Ltd.	Cambridge	144.11	1974	CCA
10	Northern Pressure Treated Wood	Kirkland Lake	124.1	1974	PCP/CCA
11	Northern Wood Preservers Inc.	Thunder Bay	719.22	1934	PCP/Creosote/CCA
12	Norwood Treatment Ltd	North Bay	28.42	1988	CCA
13	Pastway Planing Ltd	Combermere	140.16	1986	CCA
14	Ram Forest Products Inc.	Vandorf	208.16	1980	CCA
15	Shelbourne Wood Processing	Shelbourne	183.07	1985	CCA
16	South River Forest Products Ltd.	South River	24.02	1989	CCA
17	Toronto Wood Treating Ltd.	Acton	84.87	1977	CCA
18	Total Forest Industries	Hagersville	196.15	1988	CCA
19	Trent Timber Treating Inc.	Peterborough	209.76	1978	CCA
20	Trilake Timber Company Ltd.	Kenora	80.06	1960	CCA
		Total	3176.4		

(a): CCA: Chromated Copper Arsenate
PCP: Pentachlorophenol

(b) CCA retorts volume only; includes 1 PCP thermal tank and 2 thermal butt tanks.

Table 2: Estimated volume of treated wood in Ontario (1990)

Processes	Number of Plants	Retorts Volume (m ³)	Treatment Factor(a) (m ³ /m ³)	Treatment (1000 m ³)
CCA	17	2,240.4	659	1476.4
Creosote	2	433.7	129.7	56.3
PCP	4	502.3	131.8	66.2
TOTAL	20 (b)	3,176.4	--	1598.9

- (a) Treatment factor: annual volume (m³) of treated wood per volume (m³) of the retorts at the plant.
CCA Treatment Factor are calculated from audit data (Brown, 1991).
Creosote and PCP Treatment Factors are from Micklewright (1987) study.

- (b) The total number of plants is 20. Some plants use more than one process.

Table 3: Waste generation factors (L/m³, a) in wood preserving industry (b)

Processes	Wastewater	Wastewater Treatment Residuals	Process Residuals	Dripping
CCA	-	-	0.081 (c)	0.17
Creosote	174	2.68	0.542	0.17
PCP	174	2.68	0.845	0.17

(a) Unit: litre of waste generated per cubic metre of wood treated.

(b) Source: USEPA (1987), unless noted otherwise.

(c) Emission factor calculated from Environment Canada audit.

Table 4: Estimated volumes of waste generated by the Ontario wood preserving industry (a)

Processes	Production (1000 m ³)	Wastewater (kL)	Wastewater Treatment Residuals (kL)	Process Residuals (kL)	Dripping (kL)
CCA	1476.4	-	-	119.6	251
Creosote	56.3	9,796	151 (b)	30.5	9.6
PCP	66.2	11,518	177 (b)	55.9	11.3
TOTAL	1,598.9	21,314	328	206.0	271.9

(a) Calculated from the treatment volumes (Table 2) and generation factors (Table 3).

(b) Wastewater treatment residuals from PCP and creosote plants are listed in Schedule 1 of Regulation 347.

Table 5: Chemical analysis of soil samples

	Just Off The Drip Pad (ug/g)				
	Copper	Arsenic	Total Chromium	Hexavalent Chromium	% Moisture
Average	333	403	802	0.49	5.3
Standard Deviation	465	580	535	0.77	4.2
Minimum	19	3	38	0.00	2.5
Maximum	2,000	2,430	1,920	3.45	21.1
Number of Samples	17	17	17	17	17
	Off site (no obvious contamination) (ug/g)				
Average	54	28	471	0.14	9.7
Standard Deviation	44	32	433	0.37	3.9
Minimum	13	1.2	16	0.00	4.3
Maximum	193	127	1,550	1.55	18.1
Number of Samples	17	17	17	16	17
	Storage Area (ug/g)				
Average	266	2,170	738	0.30	8.6
Standard Deviation	730	11,047	1,023	0.32	7.7
Minimum	22	3.5	22	0.00	2.3
Maximum	4,220	63,632	5,600	1.20	42.4
Number of Samples	32	32	32	32	32
Decommissioning Guidelines	225	40	750	8	—

	Chemical Analysis of Process Residuals (% , unless noted)				
Average	3.5	7.8	3.3	182.1 ug/g	27.0
Standard Deviation	4.9	6.6	2.8	432.7 ug/g	19.8
Minimum	0.7	0.9	0.5	0.8 ug/g	4.7
Maximum	22.3	23.1	11.4	1,730 ug/g	70
Number of Samples	19	19	19	16	19

Source: Calculation based on data supplied by Environment Canada's Ontario Audit (Brown, 1991)

TABLE 6: Wastes from wood preservation using pentachlorophenol (a)

	Wastewaters (ppm)	Process Sludges or Residuals (ppm)	Preservatives Formulations (Dripping) (ppm)
Pentachlorophenol	0.01-310	40-34,000	14,000-52,000
Benzo(a)anthracene	0.03-10.0	5.1-2,800	75
Benzo(a)pyrene	0.007-10.0	54-1,100	50
Dibenz(a,h)- anthracene	0.1-1.0	50-310	7
Indeno(1,2,3-c,d)- pyrene	0.006-10.0	16-130	4 (b)
Arsenic	0.003-33.0	NA (c)	NA
Chromium	0.004-14.0	NA	NA
	(ppb)	(ppb)	(ppb)
TCDDs	0.001-8	0.001-5	1
PeCDDs	0.008-20	0.2-2	30-70
HxCDDs	0.03-200	0.06-5,000	100-5,000
HpCDDs	0.009-80	0.5-140,000	9,000-100,000
TCDFs	0.0006-2	0.01-35	1-30
PeCDFs	0.001-300	0.08-1,000	100-1,000
HxCDFs	0.001-10	0.01-13,000	200-10,000
HpCDFs	0.002-50	0.3-16,000	100-13,000

(a) Source: U.S. Federal Register, 1988.

(b) NA: not analyzed.

(c) Trace: concentration reported represents detection limit of the analytical method used.

TABLE 7: Inorganic wastes (a, b)

(Constituents of concern and range of detected concentration)

	Process sludge or residuals (ppm)	Unused Preservatives (ppm)
Arsenic	5,300-760,000	5,500
Chromium	70-33,000	6,200

a) Source: Federal Register, 1988.

b) Presently the USEPA does not have reliable waste characterization data for wastewaters from inorganic processes. The agency believes that the data for unused preservative are representative of both drippage and process wastewaters from inorganic processes.

TABLE 8: Creosote wastes

(Constituents of Concern and Range of Detected Concentration)

	Wastewaters (ppm)	Process Sludges or Residuals (ppm)	Unused Formulations (Dripping) (ppm)
Benzo(a)anthracene	0.03-10	280-7,500	1,600-2,600
Benzo(a)pyrene	0.007-1	1,800-3,000	400-600
Benz(k)- fluoranthene	0.02-4	2,300	21,400
Dibenz(a,h)- anthracene	0.1-1	140-680	100-400
Indeno(1,2,3-c,d)- pyrene	0.006-10	100-300	1,000
Naphthalene	0.1-400	700-64,000	13,000-18,000
Arsenic	0.003	NA	NA
Chromium	0.004-10	NA	NA

Source: U.S. Federal Register, 1988.

NA: Not Analysed

Table 9 : Summary of vector scoring

Appendix:	A	B	C	D	E
Parameter	PCP	B(a)P	TCDD	Arsenic	Chromium
1. Environmental Mobility	10	7	7	10	10
2. Environmental Persistence	10	10	10	10	10
3. Bioaccumulation	7	10	10	0	4
4. Environmental Exposure	10	10	10	10	10
5. Acute lethality	10	ND	10	8	8
6. Sublethal effects plants	8	ND	ND	6	8
7. Sublethal effects mammals	6	2	10	10	10
8. Sublethal effects non-mammals	10	ND	10	2	4
9. Teratogenicity	4	6	10	4	8
10. Mutagenicity	10	10	0	8	10
11. Carcinogenicity	2	10	10	10	10

ND: no data or insufficient data.

APPENDIX A

ENVIRONMENTAL BEHAVIOUR AND TOXICITY OF PENTACHLOROPHENOL

A.1 Chemical and physical properties of pentachlorophenol

The chemical and physical properties of pentachlorophenol are presented in Table A1.

Pentachlorophenol is one of the most widely used pesticides in Ontario. It is used primarily as an industrial wood preservative and in formulations for herbicides and pesticides. Pure pentachlorophenol is in the form of colourless crystals. Used pentachlorophenol often has the appearance of a dark grey to brown sludge or dust.

Pentachlorophenol does not occur naturally in the environment. Its presence in the environment is therefore a result of emissions in air, water or on land.

A.2 Environmental behaviour of pentachlorophenol

A.2.1 Environmental mobility

Using the Level 1 Fugacity Model (Mackay *et al.*, 1982), it is estimated that 72.8% of pentachlorophenol is found in the soil medium, 21.2% in water and 5.8% in sediment. Since the expected dispersion is in three media, the score for environmental mobility is set at 10.

A.2.2 Persistence

In soil medium, biodegradation is considered to be the major transformation mechanism for pentachlorophenol. PCP is metabolized rapidly by most acclimated microorganisms (Kaufman, 1978). Pentachlorophenol is less persistent in soils of high organic content than in soils of low organic content. High moisture content and temperature conducive to microbial activity are other factors affecting the persistence of PCP in soil (USEPA, 1986). Half-lives are usually between 2 to 4 weeks (Crospy *et al.*, 1981).

In an aquatic system, photolysis and biodegradation are the most dominant transformation processes for pentachlorophenol. On the other hand, hydrolysis and oxidation are not important mechanisms for the removal of PCP from surface waters (ATSDR, 1989).

Wong and Crosby (1981) reported that pentachlorophenol in aqueous solutions was photolyzed under laboratory UV-light irradiation with an estimated half-lives of about 100 hours at pH 3.3, and of 3.5 hours at pH 7.3. From outdoor tests conducted with river water in man-made channels, Pignatello *et al.* (1983) demonstrated that photolysis of pentachlorophenol was rapid at the water surface (half-life of 0.70 hours at 0.5 cm depth). However, photolysis was greatly attenuated with increasing depth of water column (half-life 228 hours at 30 cm depth). Photolytic degradation accounted for 5-28% decrease in the initial tests concentration of the compound after 3 weeks.

Pentachlorophenol is bio-transformed in aqueous systems by acclimated microorganisms. Liu *et al.* (1981) found that acclimated cultures of activated sludge bacteria transformed pentachlorophenol more rapidly under aerobic conditions (half-life of 0.36 days) than under anaerobic conditions (half-life of 192 days). Pignatello *et al.* (1983) reported that after about a 3-week acclimation period, microbial transformation accounted for a 26-46% decline in the initial test concentration of pentachlorophenol.

Assuming the worst case scenario under anaerobic condition in a groundwater environment, the reported half-life of 192 days would indicate that pentachlorophenol can be very persistent and consequently is scored at 10 (MOEE, 1991: i.e. half-life greater than 100 days).

A.2.3 Bioaccumulation

The log K_{ow} of 5.01 suggests that pentachlorophenol will bioaccumulate to some degree. The extent of bioaccumulation depends on the pH of the medium, since pentachlorophenol dissociates at higher ambient pH levels to the more soluble pentachlorophenate anion. Bioaccumulation of pentachlorophenol in algae, aquatic invertebrates and fish with bioconcentration factors (BCFs) of up to 10,000 have been demonstrated (ATSDR, 1989). The vector score is set at 7 for bioaccumulation.

A.2.4 Environmental exposure

Under present waste management practices (see Section 3.2 in Background Document), most of the process residuals are disposed of at a certified landfill. However, at some of the plants, drippage residuals are released into the environment by dripping on soil surface and by infiltration in groundwater.

The exposure pathway of concern is contamination of soil and groundwater. Mismanagement and improper disposal could result in substantial health and environmental adverse effects. Environmental exposure is scored at 10 for pentachlorophenol.

A.3 Toxicity of pentachlorophenol

The evaluation of toxicity of pentachlorophenol is based on the results of a preliminary assessment completed in March 1990 by a consultant for the Hazardous Contaminants Branch (MOEE, 1990). The document includes the results of literature search in TOXNET (1990), RTECS (1989), ACS (1990), and IARC (1990).

A.3.1 Acute lethality

A score of 10 is based on aquatic LC_{50} for larval carp (0.0095 mg/L), chinook salmon (0.068 mg/L), channel catfish (0.054 mg/L) and rainbow trout (0.052 mg/L) (MOEE, 1990).

Toxicity levels from other routes of exposure are scored at lower levels (MOEE, 1990). Toxicity by oral exposure is scored at 6 (LD_{50} on rat at 27 mg/kg). Toxicity by inhalation exposure is scored at 4 (LC_{50} on mouse at 225 mg/m³). And toxicity by dermal exposure is scored at 4 (LD_{50} on rat at 96 mg/kg).

Dreisbach (1980) provides an estimate of 1 g (approximately 17.0 mg/kg body weight) as the lowest human lethal dose for pentachlorophenol. St. Omer and Gadusek (1987) reported LD_{50} in the range of 50-230 mg/kg/day in rats, using technical grade pentachlorophenol.

Freshwater fish have been reported to be very sensitive to pentachlorophenol. Johnson *et al.* (1980) reported LC_{50} of 0.032 to 0.205 mg/L for bluegill, chinook salmon, rainbow trout, and fathead minnow.

A.3.2 Sublethal effects, plants

A score of 8 is based on lowest EC_{50} of 0.080 mg/L for growth inhibition of freshwater algae *Scenedesmus quadricauda* (Adema and Vink, 1981).

Due to insufficient data for terrestrial plants, a score cannot be determined for that medium (MOEE, 1990).

A.3.3 Sublethal effects, mammals

A score of 6 is based on a NOAEL (no observable adverse effect level) of 3.0 mg/kg/day of pure PCP administered orally to rats (MOEE, 1990).

Groups of male and female Sprague-Dawley rats were exposed to pentachlorophenol at dietary levels providing doses of 0, 1, 3, 10 and 30 mg/kg/day (Schwetz *et al.*, 1978). At 10 mg/kg/day and above there was significant weight increase and discoloration of the liver and kidney. At 3 mg/kg/day, there was no effects reported. Similar findings were obtained earlier by Johnson *et al.* (1973).

A.3.4 Sublethal effects, non-mammals

A score of 10 is based on an estimated MATC (maximum acceptable toxic concentration) of 0.0005 mg/L on carp (*Cyprinus carpio*) (Verma *et al.*, 1981). The score of 10 is also supported by data from a study on sockeye salmon (*Oncorhynchus nerka*) by Webb and Brett (1973): at 0.0032 mg/L there was a 10% growth inhibition and the threshold for effects on growth and food conversion efficiency was identified at 0.0018 mg/L.

A.3.5 Teratogenicity

A score of 4 is based on teratogenic effects observed on rat ingesting orally a dosage of 13 mg/kg/day (MOEE, 1990). In the study conducted by Welsh *et al.* (1987), male and female Sprague-Dawley rats were given daily PCP dosage of 0, 4, 13, or 43 mg/kg body weight, during the mating and pregnancy period lasting 181 days. The dose level of 43 mg/kg caused a decrease in the weight gain of dams and was lethal to embryos. At 13 mg/kg, growth retardation and an increased incidence of skeletal variations were observed in the fetuses.

In a similar study done earlier (Schwetz *et al.*, 1974), pregnant Sprague-Dawley rats were treated orally with both commercial and pure pentachlorophenol in corn oil. The dose levels were 5, 15, 30 and 50 mg/kg. Treatment was on days 6-15 of gestation. No anomalies were reported at the 5 mg/kg level. At levels greater than 15 mg/kg there was significant incidence of litters having soft-tissue anomalies and various types of skeletal anomalies.

A.3.6 Mutagenicity

A score of 10 is based on numerous studies showing positive evidence of mutagenicity *in vitro* and *in vivo* (MOEE, 1990).

Pentachlorophenol has been tested in several *in vitro* studies. In bacteria, it did not induce gene mutations in assays with and without a metabolic activation system (Andersen, 1972; Fahrig, 1974; Lemma, 1975; Moriya, 1983; Simmon, 1977; Waters, 1982). Positive results were reported for DNA damage in the *Bacillus subtilis* rec-assays system (Waters, 1982) using technical grade pentachlorophenol; negative results were reported in the *E. coli* polymerase A test (Waters, 1982). These results do not necessarily conflict because the two assays have different end points. In yeast, pentachlorophenol induced gene mutation (Fahrig, 1974; Fahrig, 1978) and genetic recombination (Fahrig, 1978; Waters, 1982).

Pentachlorophenol has been tested *in vivo* in animals in only one assay. It increased the number of mice with somatic mutations in the mouse spot test, and the authors of the study concluded that the increase was evidence of a weak but definite mutagenic response (Fahrig, 1978). In *Drosophila*, it did not induce sex-linked recessive mutations (Fahrig, 1974; Fahrig, 1978). It was not mutagenic in two strains of bacteria when tested in a mouse host-mediated assay system (Buserlmaier, 1973).

A.3.7 Carcinogenicity

A score of 2 is based on limited evidence of carcinogenicity in mouse via subcutaneous route. The subcutaneous administration of the dose tends to overestimate the hazard that would be found in normal exposure routes (inhalation or ingestion) (MOEE, 1990). Other studies are negative, although there is *in vitro* evidence of one genic transformation (Sax, 1988).

Pentachlorophenol is classified as a Group 3 chemical by IARC (1982).

A.4 Results of vector scoring of pentachlorophenol

The results of the vector scoring system is summarized in Table A2.

Environmental behaviours of pentachlorophenol increase significantly the risk of adverse effect on human health and the environment: the chemical is mobile in the environment where it will persist, and exposure routes are relatively available. In terms of toxicity, it is a potent lethal chemical, specially for aquatic species. It is also a known mutagen, but not a carcinogen.

A.5 References

ACS, 1990.

American Chemical Society. Chemical Abstract Registry File Database. CA Registry.

Andersen, K.J., E.G. Leighty and M.T. Takahashi, 1972.

Evaluation of herbicides for possible mutagenic properties. J. Agric. Food Chem. 20:649-659.

Adema, D.M.M. and G.J. Vink, 1981.

A comparative study of the toxicity of 1,1,2-trichloroethane, dieldrin, pentachlorophenol and 3,4-dichloroaniline for marine and freshwater organisms. Chemosphere 10(6) 533-554.

ATSDR, 1989.

Toxicological profile for pentachlorophenol. Prepared by Clement Associates for the Agency for Toxic Substances and Disease Registry (ATSDR), U.S. Public Health Service, in collaboration with USEPA. December, 1989. PB90-182163.

Buserlmaier, W., G. Rohrborn and P. Propping, 1973.

Comparative investigations on the mutagenicity of pesticides in mammalian test systems. Mutat. Res. 21:25-26.

Crosby, D.G., K.I. Beynon, P.A. Greve, F. Korte, G.G. Still and J.W. Vonk, 1981.

Environmental chemistry of pentachlorophenol. Pure Appl.Chem. 53:1051-1080.

Dreisbach, R.H., 1980.

Handbook of poisonings: Prevention, diagnosis, and treatment. 10th ed., Lange Medical Publications: Los Altos, CA, p. 364.

Fahrig, R., 1974.

Comparative mutagenicity studies with pesticides. IARC Scientific Publication 10:161-181.

Fahrig, R., C. Nilson and C. Rappe, 1978.

Genetic activity of chlorophenols and chlorophenol impurities. In: Rao KR, ed. Pentachlorophenol: Chemistry, pharmacology and environmental toxicology. New York NY: Plenum Press, 325-338.

IARC, 1982.

IARC monograph on the evaluation of the carcinogenic risk of chemicals to humans. Supplement 29, International Agency for Research on Cancer, Lyon, France.

IARC, 1990.

International Agency for Research on Cancer. 1972-1990. IARC Monographs on the evaluation of carcinogenic risk of chemicals to man. World Health Organisation, Lyon, France. Vol 41, p. 319. (1986).

Johnson, R.L., P.J. Gehring and R.J. Kociba, 1973.

Chlorinated dibenzodioxins and pentachlorophenol. Environ. Health Perspect 5:171-175.

Johnson, W.W. and M. T. Finley. 1980.

Handbook of acute toxicity of chemicals to fish and aquatic invertebrates. US Dept of Interior, Fish and Wildlife Serv. Resource Publ. No. 137.

Lemma, A. and B.N. Ames, 1975.

Screening for mutagenic activity of some molluscicides. Trans R Soc Med Hyg. 69:167-168.

Kaufman, D.D., 1978.

Degradation of pentachlorophenol in soil and by soil organisms. In: Pentachlorophenol, Chemistry, Pharmacology, and Environmental Toxicology. Rao K.R. ed., New York, NY; Plenum Press; 27-39.

Liu, D., K. Thomson and W.M.J. Strachan, 1981.

Biodegradation of pentachlorophenol in a simulated aquatic environment. Bull. Environ Contam. Toxicol. 26:85-90.

Mackay, D. and S. Paterson, 1982.

Fugacity revisited. Env. Sc. and Tech., Vol.16, No.12.

MOEE, 1990.

Pentachlorophenol. Ontario environmental assessment: preliminary assessment. Prepared for the Hazardous Contaminants Branch, March, 1990.

MOEE, 1991.

Guidance manual for hazardous waste categorization and review program. Prepared by Waste Management Branch, Ontario Ministry of Environment and Energy (MOEE), January 1991.

Moriya, M. T. Ohta and K. Watanabe, 1983.

Further mutagenicity studies on pesticides in bacterial reversion assay systems. Mutat Res 116:185-216.

- Pignatello, J.J., M.M. Martison and J.G. Steiert, 1983.
Biodegradation and photolysis of pentachlorophenol in artificial freshwater streams. Appl. Environ. Microbiol. 46:1024-1031.
- RTECS, 1989.
Registry of Toxic Effects of Chemical Substances. Computerized database produced by the National Institute of Occupational Safety and Health (NIOSH), provided by the Canadian Centre for Occupational Health and Safety. Updated June, 1989.
- Sax, N.I., 1988.
Dangerous properties of industrial materials. 7th Edition. Van Nostrand Reinhold Co., New York, NY.
- Schwetz, B.A., P.A. Keeler and P.J. Gehrins, 1974.
The effect of purified and commercial grade pentachlorophenol on rat embryonal and fetal development. Toxicol. Applied Pharmacol., 28:151-161.
- Schwetz, B.A., J.F. Quast and P.A. Keeler, 1978.
Results of two year toxicity and reproduction studies on pentachlorophenol In: Rao KR, ed. Chemistry, pharmacology and environmental toxicology. New York NY: Plenum Press, 301-309.
- Simmon, V.F., A.D. Mitchell and T.A. Jorgenson, 1977.
Evaluation of selected pesticides as chemical mutagens. In vitro and in vivo studies. U.S. Environmental Protection agency. EPA 600/1-77-028.
- St. Omer, V.E.V. and F. Gadusek, 1987.
The acute oral LD₅₀ of technical pentachlorophenol in developing rats. Environ. Toxicol. Chem 6:147-149.
- TOXNET, 1990.
Toxicology Data Network. National Library of Medicine, U.S. Department of Health and Human Services. Computerized database searched on January 15, 1990.
- USEPA, 1986.
Health and environmental effects profile for pentachlorophenol. June 1986. Report No. EPA/600/X-86/170. PB89-124473
- Verma, S.R., I.P. Tonk and R.C. Dalela, 1981.
Determination of the maximum acceptable toxicant concentration (MATC) and the safe concentration for certain aquatic pollutants. Acta Hydrochim. Hydrobiol. 9(3): 247-254.

- Waters, M.D., S.S. Sandu and V.F. Simmon, 1982.
Study of pesticide genotoxicity. *Basic Life Sci.*, 21:275-326.
- Webb, P.W. and J.R. Brett, 1973.
Effects of sublethal concentrations of sodium pentachlorophenate on growth rate, food conversion efficiency, and swimming performance in underyearling sockeye salmon (*Oncorhynchus nerka*). *J. Fish. Res. Board Can.* 30:499.
- Welsh, J.J., T.F.X. Collins, T.N. Black, S.L. Graham and M.W. O'Donnell Jr., 1987.
Teratogenic potential of purified pentachlorophenol and pentachloroanisole in subchronically exposed Sprague-Dawley rats. *Fd. Chem. Toxic.* 25:163-172.
- Wong, A.S. and D.G. Crosby, 1981.
Photodecomposition of pentachlorophenol in water. *J Agric. Food Chem* 29:130-135.

Table A1: Properties of Pentachlorophenol

Chemical Abstract #	: 87-86-5
Chemical Abstract Name	: Pentachlorophenol
Synonyms	: 2,3,4,5,6-Pentachlorophenol PCP, Pencilorol
Trade Names	: Chlorophen Penta Cryptogyl 01 PCP Dowicide 7 Santobrite Dowicide G Santophen 20 Pentchlorol Witophen P
Chemical Formula	: C_6HCl_5O
Molecular Weight	: 266.35
Boiling Point	: 310 °C
Melting Point	: 190-191°C (anhydrous)
Density	: 1.978 g/cm ³ (22°C)
Solubility	: 14 mg/L (20°C)
Volatility	: 0.00011 mm Hg at 20 °C
Flash Point (open cup)	: not flammable
log octanol/water (partition coefficient)	: 5.01

Table A2: Results of vector scoring of pentachlorophenol

<u>Parameter</u>	<u>Score</u>	<u>Concern Levels (a)</u>
1. Environmental Mobility	10	≥ 7
2. Environmental Persistence	10	≥ 7
3. Bioaccumulation	7	≥ 7
4. Environmental Exposure	10	≥ 7
5. Acute lethality	10	≥ 6
6. Sublethal effects plants	8	≥ 4
7. Sublethal effects mammals	6	≥ 4
8. Sublethal effects non-mammals	10	≥ 4
9. Teratogenicity	4	≥ 2
10. Mutagenicity	10	≥ 6
11. Carcinogenicity	2	≥ 2

(a): concern levels established for the listing of hazardous wastes.

APPENDIX B

ENVIRONMENTAL BEHAVIOUR AND TOXICITY OF BENZO(A)PYRENE

B.1 Chemical and physical properties of benzo(a)pyrene

Benzo(a)pyrene (B(a)P) is a polycyclic aromatic hydrocarbon (PAH), formed during the incomplete burning of fossil fuel, domestic waste or any organic matter. B(a)P is also a component of coal tar used in different industries (e.g. road paving, roofing) and a component in creosote used in the wood preserving industry (ATSDR, 1990).

Selected chemical and physical properties of B(a)P are presented in Table B1.

B.2 Environmental behaviour of benzo(a)pyrene

B.2.1 Environmental mobility

Using the Level 1 Fugacity Model (Mackay *et al.*, 1982), it is estimated that 86% of B(a)P is found in the soil medium, 7% in sediment and 6% in water.

Since the model indicates that one medium contains more than 80% of the contaminant, the environmental mobility of benzo(a)pyrene is scored at 7 (MOEE, 1991).

B.2.2 Persistence

In terrestrial systems, the important fate process is biodegradation. Generally, biodegradation processes are very slow. Bossert and Bartha (1986) reported that 72% of the original amount of B(a)P in soil existed after 16 months of incubation with bacteria. Coover and Sims (1987) estimated a half-life of 290 days for B(A)p in soil.

In sediment and aquatic systems, biodegradation is also the most important fate process. A half-life of 2.4 years has been reported for B(a)P (USEPA, 1979). In contaminated water streams, half-lives for B(a)P range from 10 to 400 times longer. These long half-lives indicate that benzo(a)pyrene is relatively persistent in sediment and aquatic systems.

Based on these figures, persistence of benzo(a)pyrene is scored at 10 (MOEE, 1991).

B.2.3 Bioaccumulation

Based on a Log K_{ow} value of 6.04 (Montgomery *et al.*, 1990), bioaccumulation is scored at 10.

B.2.4 Environmental exposure

Under present waste management practices (see Section 3.2 in Background Document), most of the process residuals from wood preservation are disposed of at a certified landfill. However, at some of the plants, drippage residuals are released into the environment by dripping on soil surface and infiltration in groundwater.

The exposure pathway of concern is contamination of groundwater. Mismanagement and improper disposal could result in substantial health and environmental adverse effects. Environmental exposure is scored at 10.

B.3 Toxicity of benzo(a)pyrene

The evaluation of toxicity of benzo(a)pyrene is based on the results of a preliminary assessment completed in February 1992 (MOEE, 1992). A comprehensive toxicological profile on B(A)P has been completed recently (ATSDR, 1990).

B.3.1 Acute lethality

From a recent toxicological profile prepared by ATSDR (1990), there is no pertinent data on acute lethality either on human or on experimental animals, using oral, dermal or inhalation exposure routes.

The acute lethality of B(a)P has been investigated however by Salamone (1981). The LD₅₀ for B(a)P administered intraperitoneally to male mice was determined at approximately 250 mg/kg.

Acute lethality cannot be scored for benzo(a)pyrene due to lack of data.

B.3.2 Sublethal effects, plants

Data on the sublethal effects on plants could not be found in the available literature.

B.3.3 Sublethal effects, mammals

Decreased longevity have been reported in "nonresponsive" strains of mice following subchronic oral exposure to 120 mg/kg. The death was due to haemorrhage or infection in "responsive" mice, following a single intraperitoneal dose of 500 mg/kg (Robinson *et al.*, 1975).

Sublethal effects on mammals are scored at 2 for benzo(a)pyrene.

B.3.4 Sublethal effects, non-mammals

Data on the sublethal effects on non-mammals could not be found in the available literature.

B.3.5 Teratogenicity

Placental transfer of benzo(a)pyrene has been observed in mice (Shendrikova *et al.*, 1974; Shendrikova and Aleksandrov, 1974) following intravenous injection and oral administration. The results of two oral studies in mice (Mackenzie and Angevine 1981, Legraverend *et al.*, 1984) and one in rats (Rigdon and Rennels, 1964) indicate that in utero exposure to benzo(a)pyrene is associated with developmental toxicity and adverse reproductive effects. Adverse developmental / reproductive effects resulted from oral exposure to doses of benzo(a)pyrene of 10 mg/kg/day (Mackenzie and Angevine, 1981). Investigations by Legraverend *et al.* (1984) suggest that it is benzo(a)pyrene and not a metabolite of benzo(a)pyrene which is responsible for these adverse effects.

No data were available in experimental animals regarding the reproductive and developmental effects of benzo(a)pyrene following inhalation or dermal exposures. However, adverse developmental and reproductive effects were observed in several injection studies. Adverse effects observed following intraperitoneal injection of benzo(a)pyrene in mice included: stillbirths, resorption, and malformations (Shum *et al.*, 1979; Hoshino *et al.*, 1981); decreases in follicular growth and corpora lutea (Swartz and Mattison, 1985; Payne, 1958); immunosuppression (Urso and Gengozian, 1980); and tumor induction (Bulay and Wattenberg, 1971).

Adverse effects, such as tumor induction in mice (Nikonova, 1977), were observed following subcutaneous injection of benzo(a)pyrene. Decreased fetal survival was reported in Swiss mice following direct embryonal injection of benzo(a)pyrene (Barbieri *et al.*, 1986).

Sheveleva (1978) administered benzo(a)pyrene by gavage to rats at doses of 0, 0.05, 0.5, and 5 mg/kg/day on days 1-15, 3-4, and 9-10 of pregnancy. The dams receiving 0.5 and 5 mg/kg on days 1-15 showed signs of maternal toxicity including decreased weight gain and haematological changes.

Teratogenicity is scored at 6 for B(a)P.

B.3.6 Mutagenicity

Benzo(a)pyrene shows positive mutagenic activity *in vitro* in several strains of *Salmonella typhimurium* in the presence of either rodent microsomes or hepatocytes for exogenous metabolic activation. Negative results were reported for host-mediated activation (Glatt *et al.*, 1985; Simmon *et al.*, 1979). Body fluids from rodents exposed *in vivo* to benzo(a)pyrene showed positive mutagenic activity in three strains of *Salmonella typhimurium* (Batzinger *et al.*, 1978; Conner *et al.*, 1979). Generally, benzo(a)pyrene shows positive genotoxic activity *in vivo* mammalian cell systems with either exogenous or endogenous metabolic activation.

There is sufficient evidence from short term *in vivo* and *in vitro* genetic toxicology tests to prove that benzo(a)pyrene is a potent genotoxic agent when metabolically activated. There is sufficient evidence that benzo(a)pyrene interact with mammalian gonads or germ cell DNA and that it induces such end points as unscheduled DNA synthesis (Sega, 1979), chromosomal aberrations (Basler and Rohrborn, 1978), and morphological abnormalities (Wyrobek *et al.*, 1981).

Mutagenicity is scored at 10 for benzo(a)pyrene.

B.3.7 Carcinogenicity

The most sensitive (occurs at the lowest doses) and well-studied end point of benzo(a)pyrene-induced intermediate and long term toxicity is cancer. Benzo(a)pyrene has been found to cause cancer in laboratory animals by all routes of exposure by which humans can be expected to be exposed (inhalation, oral, dermal). In addition, benzo(a)pyrene has elicited tumors in animals using a number of experimental routes of exposure such as intraperitoneal, intrapulmonary, and subcutaneous injections, as well as transplacentally (IARC, 1973; 1983).

A series of steps is involved between exposure to benzo(a)pyrene and tumor development; these involve metabolic activation and genotoxicity. Target tissues for benzo(a)pyrene-induced carcinogenesis possess the ability to metabolically activate benzo(a)pyrene to its reactive epoxide derivative. Covalent binding of reactive metabolites of benzo(a)pyrene to DNA can occur and result in the formation of DNA adducts. This step appears to be essential for the production of benzo(a)pyrene-induced neoplasia (Gelboin, 1980; Weinstein *et al.*, 1978).

B(a)P is classified as a Group 2A chemical by IARC (1982). Based on the information above, carcinogenicity is scored at 10 for benzo(a)pyrene.

B.4 Results of Vector Scoring System of benzo(a)pyrene

The vector scores, as shown in Table B2, indicate that benzo(a)pyrene is of high concern because of its teratogenicity, mutagenicity, and carcinogenicity. It is also of concern because of its persistence in the environment and ability to bioaccumulate.

B.5 References

ATSDR, 1990.

Toxicological profile for benzo(a)pyrene. Prepared by ICF-Clement, for the Agency for Toxic Substances and Disease Registry (ATSDR), U.S. Public Health Service, in collaboration with USEPA. May 1990. ATSDR/TP-88/05. PB90-258245.

Barbieri, O., E. Ognio, O. Rossi, S. Astigiano, and L. Rossi, 1986.

Embryotoxicity of benzo(a)pyrene and some of its synthetic derivatives in swiss mice. *Cancer Res.* 46:94-98.

Basler, A., and G. Rohrborn, 1978.

Mutagenicity of polycyclic hydrocarbons. IV. Correlated studies with anthracene, benzo(a)anthracene, benzo(a)pyrene, chrysene and phenanthrene. *Proc. Perugia Qudrenn.Int. Conf. Cancer.* 6:843-849.

Batzinger, R.P., S.L. Ou, and E. Bueding, 1978.

Antimutagenic effects of 2(3)-tert-butyl-4-hydroxyanisole and of antimicrobial agents. *Cancer Res.* 38:4478-4485.

Bossert, I.D., and R. Bartha, 1986.

Structure biodegradability relationships of polycyclic aromatic hydrocarbons in soil. *Bull. Environ. Contam. Toxicol.* 37:490-495.

Bulay, O.M. and L.W. Wattenberg, 1971.

Carcinogenic effects of polycyclic hydrocarbon carcinogen administration to mice during pregnancy on the progeny. *J. Nat. Cancer. Inst.* 46:397-402.

Conner, T.H., G.C. Forti, P. Sitra and M.S. Legator, 1979.

Bile as a source of mutagenic metabolites produced vivo and detected by *Salmonella typhimurium*. *Environ. Mut.* 1:269-276.

Coover, M.P. and R.C. Sims, 1987.

The effect of temperature on polycyclic aromatic hydrocarbons persistence in an unacclimated agricultural soil. *Haz. Waste AND Haz. Mat.* 4:69-82.

Gelboin, H.W., 1980.

Benzo(a)pyrene metabolism, activation and related enzymes. *Physiol. Rev.* 60:1107-1166.

Glatt, H., M. Buecker, K.L. Platt and F. Oesch, 1985.

Host-mediated mutagenicity experiments with benzo(a)pyrene and two of its metabolites. *Mutat. Res.* 156:163-169.

- Hoshino, K., Y. Hayashi, Y. Takehira and Y. Kameyama, 1981.
Influences of genetic factors on the teratogenicity of environmental pollutants: teratogenic susceptibility to benzo(a)pyrene and Ah locus in mice. *Cong. Anom.* 21:97-103.
- IARC, 1973.
IARC (International Agency for Research on Cancer) monographs on the evaluation of carcinogenic risk of chemical to man. Volume 3: certain polycyclic aromatic hydrocarbons and heterocyclic compounds. IARC, Lyon, France.
- IARC, 1982.
IARC monograph on the evaluation of the carcinogenic risk of chemicals to humans. Supplement 29, International Agency for Research on Cancer, Lyon, France.
- IARC, 1983.
IARC monographs on the evaluation of carcinogenic risk of chemical to humans. IARC, Lyon, France. Vol. 32.
- Legraverend, C., T.M. Guenther and D.W. Nebert, 1984.
Importance of the route of administration for genetic differences in benzo(a)pyrene induced in utero toxicity and teratogenicity. *Teratology.* 29:333-35-47.
- Mackay, D. and S. Paterson, 1982.
Fugacity revisited. *Env. Sc. and Tech.*, Vol.16, No.12, 1982.
- Mackenzie, K.M. and D.M. Angevine, 1981.
Infertility in mice exposed in utero to benzo(a)pyrene. *Biol. Reproduc.* 24:183-191.
- MOEE, 1991.
Guidance manual for hazardous waste categorization and review program. Prepared by Waste Management Branch, Ontario Ministry of Environment and Energy (MOEE), January 1991.
- MOEE, 1992.
Ontario environmental assessment: preliminary assessment of benzo(a)pyrene. Prepared by the Hazardous Contaminants Branch, Ontario Ministry of Environment and Energy (MOEE), March 1992.
- Montgomery, J.H. and L.M. Welkom, 1990.
Groundwater chemicals desk reference. Lewis Publishers, Chelsea, MI.
- Nikonova, T.V., 1977.
Transplacental action of benzo(a)pyrene and pyrene. *Bull. Exp. Biol. Med.* 84:1025-1027.

- Payne, S., 1958.
The pathological effects of the intraperitoneal injection of 3,4-benzopyrene into rats and mice. Brit. J. Cancer. 12:65-74.
- Rigdon, R.H. and G. Rennels, 1964.
Effect of feeding benzo(a)pyrene on reproduction in the rat. Experientia. 20:224-226.
- Robinson, J.R., J.S. Felton, R.C. Levitt, S.S. Thorgeirson and D.W. Nebert, 1975.
Relationship between "aromatic hydrocarbon responsiveness" and the survival times in mice treated with various drugs and environmental compounds. Mol. Pharm. 11:850-865.
- Salamone, M.F., 1981.
Toxicity of 41 carcinogens and noncarcinogenic analogs. In: Evaluation of short-term tests for carcinogens: report of the International Collaborative Program. Prog. Mutat. Res. 1:686-697.
- Sega, G.A., 1979.
Unscheduled DNA synthesis (DNA repair) in the germ cells of male mice: its role in the study of mammalian mutagenesis. Genetics. 92:S49-S58.
- Shendrikova, I.A. and V.A. Aleksandrov, 1974.
Comparative penetration of polycyclic hydrocarbons through rat placenta into the fetus. Bull. Exp. Biol. Med. (USSR). 77:169-171.
- Shendrikova, I.A., M.N. Ivanov-Golitsyn and A.Y. Likchachev, 1974.
The transplacental penetration of benzo(a)pyrene in mice. Voprosy Onkologii. 20:53-56.
- Sheveleva, G.A., 1978.
On the effect of 3,4-benzopyrene on the development of the foetus applied at different stages of gestation. Gigiena Truda I Professional nye Zabolevaniya. 7:54.
- Shum, S., N.M. Jensen and D.W. Nebert, 1979.
The murine Ah locus: in utero toxicity and teratogenesis associated with genetic differences in benzo(a)pyrene metabolism. Teratology. 20:365-376.
- Simmon, V.F., 1979.
In vitro mutagenicity of chemical carcinogens and related compounds with *Salmonella typhimurium*. J. Natl. Cancer Inst. 62(4):893-899.
- Swartz, W.J. and D.R. Mattison, 1985.
Benzo(a)pyrene inhibits ovulation in C57BL/6N mice. Anatomical Record. 212:268-276.

Urso, P. and N. Gengozian, 1980.

Depressed humoral immunity and increased tumor incidence in mice following in utero exposure to benzo(a)pyrene. *J. Toxicol. and Environ. Health.* 6:569-576.

USEPA, 1979.

Water-related environmental fate of 129 priority pollutants. U.S. Environmental Protection Agency, Washington, D.C., December 1979. EPA 440/4-79-029.

Weinstein, I.B., A.M. Jeffrey, S. Leffler, P. Pulkrabek, H. Yamasaki and D. Grunberger, 1978.

Interaction between polycyclic aromatic hydrocarbons and cellular macromolecules. In Ts'ao, P.O.P., and Gelboin, H.V. (eds.). *Polycyclic hydrocarbons and Cancer. Vol. 2: Molecular and Cell Biology.* Academic Press, New York. pp 3-36.

Wyrobek, A., L. Gordon, and G. Watchmaker, 1981.

Effect of 17 chemical agents including 6 carcinogen/noncarcinogen pairs on sperm shape abnormalities in mice. In: *Evaluation of short-term tests for carcinogens: report of the International Collaborative Program.* *Prog. Mutat. Res.* 1:712-717.

Table B1: Properties of Benzo(a)pyrene

Chemical Abstract #	: 50-32-8
Chemical Abstract Name	: benzo(a)pyrene
Synonyms	: Benzo(def)chrysene 3,4-benzopyrene 3,4-benzpyrene benz(a)pyrene BP B(a)P
Chemical Formula	: $C_{20}H_{12}$
Molecular Weight	: 252.3
Partition coefficient (log K_{ow})	: 6.04
Boiling Point	: 495 °C
Melting Point	: 179.3 °C
Density	: 1.351 g/cc.
Solubility	: 3.8 ug/L
Vapour pressure (25°C)	: 5.6×10^{-6} mm Hg
Flash Point (open cup)	: Unknown
Conversion Factor (in air)	: 1 ppm = 10.32 mg/m ³

Table B2: Results of Vector Scoring of benzo(a)pyrene

<u>Parameter</u>	<u>Score</u>	<u>Concern Levels (a)</u>
1. Environmental Mobility	7	≥ 7
2. Environmental Persistence	10	≥ 7
3. Bioaccumulation	10	≥ 7
4. Environmental Exposure	10	≥ 7
5. Acute lethality	ND ^b	≥ 6
6. Sublethal effects plants	ND ^b	≥ 4
7. Sublethal effects mammals	2	≥ 4
8. Sublethal effects non-mammals	ND ^b	≥ 4
9. Teratogenicity	6	≥ 2
10. Mutagenicity	10	≥ 6
11. Carcinogenicity	10	≥ 2

(a): concern levels established for the listing of hazardous wastes.

(b): ND = no data, or insufficient data.

APPENDIX C

ENVIRONMENTAL BEHAVIOUR AND TOXICITY OF 2,3,7,8-TCDD

C.1 Chemical and physical properties of dioxin

The toxicity of dioxin and furan mixtures is usually expressed in terms of "toxicity equivalency factors" (TEF). Since there are some 210 forms of dioxin and furan, the individual toxicity of each form is compared with the most toxic form, i.e. 2,3,7,8-tetrachlorodibenzo-p-dioxin. The equivalency factor for a specific form of dioxin or furan is therefore a fraction representing the toxicity relative to the toxicity of 2,3,7,8-TCDD. The following are TEF for some of the dioxins and furans (Environment Canada, 1990):

<u>Contaminants</u>	<u>TEF</u>
2,3,7,8-tetrachlorodibenzodioxin	1
1,2,3,7,8-pentachlorodibenzodioxin	0.5
1,2,3,4,7,8-hexachlorodibenzodioxin	0.1
1,2,3,4,7,9-hexachlorodibenzodioxin	0.1
1,2,3,6,7,8-hexachlorodibenzodioxin	0.1
1,2,3,4,6,7,8-heptachlorodibenzodioxin	0.01
Octachlorodibenzodioxin	0.001
2,3,7,8-tetrachlorodibenzofuran	0.1
2,3,4,7,8-pentachlorodibenzofuran	0.5
1,2,3,7,8-pentachlorodibenzofuran	0.05
1,2,3,4,7,8-hexachlorodibenzofuran	0.1
1,2,3,7,8,9-hexachlorodibenzofuran	0.1
1,2,3,6,7,8-hexachlorodibenzofuran	0.1
2,3,4,6,7,8-hexachlorodibenzofuran	0.1
1,2,3,4,6,7,8-heptachlorodibenzofuran	0.01
1,2,3,4,7,8,9-heptachlorodibenzofuran	0.01
Octachlorodibenzofuran	0.001

Selected chemical and physical properties of 2,3,7,8-TCDD are presented in Table C1. 2,3,7,8-TCDD, commonly called dioxin, is a colorless solid with no distinguishable odour. It does not occur naturally. It is not manufactured intentionally by any industry, but is produced in very small amounts as an impurity during the manufacture of certain herbicides and pesticides, and during incineration of municipal and industrial wastes (ATSDR, 1989).

C.2 Environmental behaviour of 2,3,7,8-TCDD

C.2.1 Environmental Mobility

Using the Fugacity Model (Level 1) (Mackay *et al.*, 1982), it is estimated that 44.0% of 2,3,7,8-TCDD is found in soil, and 50.0% in air. Since the expected dispersion is in two media, the score for environmental mobility is set at 7 (MOEE, 1991).

C.2.2 Persistence

2,3,7,8-TCDD is considered to be very persistent, because of its long half-life in lake sediment or in soil (MOEE, 1986; 1989).

Persistence of TCDD in lake sediment and water was studied under laboratory conditions (Ward *et al.*, 1978): dioxin concentrations between 0.71 and 1.83 ppm in lake sediment have a half-life near 600 days. Dioxin concentrations in water only have similar persistence, although shorter by some 10 days.

Using the fugacity model approach, the persistence of TCDD is reported at between 1 to 2 years using advection, and 12 years without advection (Mackay *et al.*, 1985).

Based on this information, environmental persistence is scored at 10.

C.2.3 Bioaccumulation

Based on a log K_{ow} of 7.9 (MOEE, 1985) and a bioconcentration factor (BCF) of 47,000 for snails (Verschuere, 1983), the score is set at 10 for bioaccumulation (MOEE, 1989; 1991).

Another study measured BCF levels in the same range: 29,200 in fathead minnow (*Pimephales promelas*) (Adams *et al.*, 1986),

C.2.4 Environmental exposure

Under present waste management practices (see Section 3.2 in Background Document), most of the process residuals from wood preservation are disposed of at a certified landfill. However, at some of the plants, drippage residuals are released into the environment by dripping on soil surface and infiltration in groundwater.

The exposure pathway of concern is contamination of surface and ground water. Mismanagement and improper disposal could result in substantial health and environmental adverse effects. Environmental exposure is scored at 10.

C.3 Toxicity of 2,3,7,8-TCDD

The evaluation of toxicity of TCDD is based on the results of a preliminary assessment completed in December 1989 by the Hazardous Contaminants Branch (MOEE, 1989). The assessment includes the results of the ATSDR (1989) toxicological profile.

C.3.1 Acute lethality

Numerous acute studies confirm the high oral toxicity of TCDD: LD₅₀ less than 0.272 mg/kg for rat, rabbit, mouse or guinea pig (MOEE, 1989; 1985). The most sensitive mammal appears to be the guinea pig: a LD₅₀ of 0.0006 mg/kg was reported in male guinea pig (Schwetz *et al.*, 1973).

Based on these results, acute lethality is scored at 10.

C.3.2 Sublethal effects, plants

Limited evidence suggests little or no phytotoxicity (MOEE, 1989; 1985). In a study conducted by Isensee (1978), no effect on algae (*Oedogonium cardiacum*) was apparent after 32 days in water containing 1.33 ug/L TCDD. In another study, pond weeds (*Elodea nuttali* and *Ceratophyllum demersum*) showed no adverse effect after several months in water containing 0.0537 ug/L TCDD.

Sublethal effects of TCDD on plants cannot be scored because of insufficient information.

C.3.3 Sublethal effects, mammals

The values of NOAEL found in studies are all below the score criterion (10) of 0.1 mg/kg. Kociba *et al.* (1978) measured a NOAEL for survival rate and tumour frequency at 0.001 ug/kg, after a 104 week experiment.

Sublethal effects on mammals is scored at 10 (MOEE, 1989).

C.3.4 Sublethal effects, non-mammals

A score of 10 is based on NOAEL results from studies on bird and mosquito larvae (MOEE, 1989; 1985).

Schwetz *et al.* (1973) found that leghorn chicks showed no adverse effect from a total oral dose (over 21 days) of 0.002 mg/kg. There were visible effects however at dose of 0.02 mg/kg.

Miller *et al.* (1973) studied mosquito larvae (*Aedes aegypti*) pulmonate snails (*Physa sp.*) and aquatic adult Oligochaete worms (*Paranais sp.*) exposed to 0.0002 mg/L TCDD over a 17-day period. There was no significant difference in the total pupation of the mosquitos, and no significant difference in survival for adult snails.

C.3.5 Teratogenicity

A score of 10 is based on numerous studies on mice exhibiting cleft palates, and on chick embryo showing cardiovascular defects when exposed to TCDD doses ranging from 0.001 to 0.100 mg/kg/day (MOEE, 1989; 1985).

C.3.6 Mutagenicity

Results of testing TCDD in mammalian systems do not provide sufficient evidence of chromosomal damaging activity for *in vivo* systems (MOEE, 1985). While a weak induction of chromosomal aberrations in rats (Green *et al.*, 1977) and induction in humans (DiLernia *et al.*, 1982) have been reported in isolated studies, the predominant evidence suggests that the compound is not clastogenic.

Mutagenicity of 2,3,7,8-TCDD is scored at 0, due to lack of evidence of mutagenic or genotoxic effects in a comprehensive battery of test systems (MOEE, 1991).

C.3.7 Carcinogenicity

A score of 10 is based on evidence of increased incidence of tumors in rats and mice, with little evidence of interaction with genetic material (MOEE, 1989). Under the IARC classification, TCDD compounds are classified in Group 2B (IARC, 1982).

The evidence is based on a comprehensive study done by Van Miller *et al.* (1977) on male Sprague-Dawley rats. In the study, 10 groups of 10 rats each were chronically exposed for 78 weeks to feed containing TCDD ranging from 1 ppt to 1,000 ppm. The highest 3 exposure levels (50, 500 and 1,000 ppm) were toxic to all animals after 4 weeks. Although the study had some shortcomings (MOEE, 1985), the study indicates an association between TCDD and induced neoplasms in rats.

C.4 Results of Vector Scoring System of Dioxins

The vector scores as indicated in Table C2 indicates that 2,3,7,8-TCDD is of high concern in the majority of the parameters examined. Except for its inconclusive effects on plants and the absence of evidence for mutagenicity, 2,3,7,8-TCDD is a potent lethal chemical that is persistent and bioaccumulate in biological systems. It is also teratogenic and a known carcinogen on animals.

C.5 References

- Adams, W.J., G.M. DeGraeve, T.D. Sabourin, J.D. Cooney and G. M. Mosher, 1986.
Toxicity and bioconcentration of 2,3,7,8-TCDD to fathead minnows (*Pimephales promelas*). Chemosphere, 15 (9-12):1503-1511.
- ATSDR, 1989.
Toxicological profile for 2,3,7,8-tetrachlorodibenzo-p-dioxin. Prepared by Syracuse Research Corporation for Agency for Toxic Substances and Disease Registry (ATSDR), U.S. Public Health Service, in collaboration with USEPA, June 1989. ATSDR/TP-88/23. PB89-214522.
- DiLernia, R., C. Crimauddo and G. Pacchetti, 1982.
The study of x-rays and TCDD effects on satellite associations may suggest a simple model for application in environmental mutagenesis. Hum. Genet. 61(1):42-47.
- Environment Canada, 1990.
Priority substances list assessment report No.1: Polychlorinated dibenzodioxins and polychlorinated dibenzofurans. Prepared by Environment Canada and Health and Welfare Canada. ISBN 0-662-17644-8. DSS cat. no. En40-215/1E.
- Green, S., F.S. Moreland and C. Sheu, 1977.
Cytogenetic effect of 2,3,7,8-tetrachlorodibenzo-p-dioxin on rat bone marrow cells. U.S. FDA, Washington, D.C., FDA By-Lines 6:292.
- IARC, 1982.
IARC monograph on the evaluation of the carcinogenic risk of chemicals to humans. Supplement 29, International Agency for Research on Cancer, Lyon, France.
- Isensee, A.R., 1978.
Bioaccumulation of 2,3,7,8-tetrachlorodibenzo-para-dioxin. In Chlorinated phenoxy acids and their dioxins, C. Ramel (ed.). Ecological Bulletin No. 27. Swedish Natural Science Research Council. pp. 255-262.
- Kociba, R.J., D.G. Keyes, J.E. Beyer, R.M. Carreon, C.E. Wade, D.A. Dittenber, R.P. Kalnins, L.E. Frauson, C.N. Park, S.D. Barnard, R.A. Hummel and C.G. Humiston, 1978.
Results of a two year chronic toxicity and oncogenicity study of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) in rats. Toxicol. Appl. Pharmacol. 46:279-303.
- Mackay, D. and S. Paterson, 1982.
Fugacity revisited. Env. Sc. and Tech., Vol.16, No.12, 1982.

- Mackay, D., S. Paterson and B. Cheung, 1985.
Evaluating the environmental fate of chemicals using the fugacity-level III approach as applied to 2,3,7,8-TCDD. *Chemosphere*; 14(6-7):859.
- Miller, R.A., L.A. Norris and C.L. Hawkes, 1973.
Toxicity of 2,3,7,8-tetrachlorodibenzo-p-dioxin (TCDD) in aquatic organisms. *Env. Health Pers.* 5:177-186.
- MOEE, 1985.
Scientific criteria document for standard development No. 4-84: PCDDs and PCDFs. Prepared for Intergovernmental Relations and Hazardous Contaminants Branch, MOEE, September 1985.
- MOEE, 1986.
CESARS (Chemical Evaluation Search and Retrieval System) document on 2,3,7,8-TCDD produced by the Ontario Ministry of Environment and Energy, and Michigan Department of Natural Resources, provided by the Canadian Centre for Occupational Health and Safety.
- MOEE, 1989.
Ontario environmental assessment: preliminary assessment of 2,3,7,8-TCDD. Produced by Ontario Ministry of the Environment and Michigan Department of Natural Resources, provided by the Canadian Centre for Occupational Health and Safety. December 1989.
- MOEE, 1991.
Guidance manual for hazardous waste categorization and review program. Prepared by Waste Management Branch, Ontario Ministry of Environment and Energy. January 1991.
- Schwetz, B.A., J.M. Norris, G.L. Sparschu, V.K. Rowe, P.J. Gehring, J.L. Emerson and C.G. Gerbig, 1973.
Toxicology of chlorinated dibenzo-p-dioxins. *Env. Health Persp.* 5:87-99.
- USEPA, 1985.
Health assessment document for polychlorinated dibenzo-p-dioxin. EPA Report No. 600/X-84-194-I. Environmental Criteria and Assessment Office, EPA Cincinnati, OH.
- Van Miller, J.P., J.J. Lalich and J.R. Allen, 1977.
Increased incidence of neoplasms in rats exposed to low levels of 2,3,7,8-tetrachlorodibenzo-p-dioxin. *Chemosphere* 6:625-632.

Verschuere, K., 1983.

Handbook of environmental data on organic chemicals. 2nd ed. Van Nostrand
Reinhold Company, New York, NY.

Ward, C.T. and F. Matsumura, 1978.

Fate of 2,3,7,8-tetrachloro dibenzo-p-dioxin (TCDD) in a model aquatic environment.
Arch. Environ. Contam. Toxicol. 7:349-357.

Table C1: Properties of 2,3,7,8-TCDD

Chemical Abstract #:	1746-01-6
Chemical Abstract Name:	2,3,7,8-tetrachloro-dibenzo-dioxin; 2,3,7,8-tetrachloro[b,e](1,4)-dioxin; 2,3,7,8-tetrachloro-p-dioxin; dioxin; 2,3,7,8-tetrachloro-1,4-dioxin; TCDBD; 2,3,7,8-TCDD.
Trade Names:	none (the compound is not produced commercially).
Chemical Formula:	$C_{12}H_4Cl_4O_2$
Molecular Weight:	321.97
Boiling Point:	412.2 °C
Melting Point:	305 °C
Density:	1.827 g/cm ³
Solubility:	7.91 ng/L (20-22 °C)
Volatility:	1.4×10^{-9} mm Hg (25 °C)
Flash Point (open cup):	Unknown
Conversion Factor:	1 ppb = 13.384 ug/m ³ at 20 °C (vapor)

Table C2: Results of vector scoring of 2,3,7,8-TCDD

<u>Parameter</u>	<u>Score</u>	<u>Concern Levels (a)</u>
1. Environmental Mobility	7	≥ 7
2. Environmental Persistence	10	≥ 7
3. Bioaccumulation	10	≥ 7
4. Environmental Exposure	10	≥ 7
5. Acute lethality	10	≥ 6
6. Sublethal effects plants	* (b)	≥ 4
7. Sublethal effects mammals	10	≥ 4
8. Sublethal effects non-mammals	10	≥ 4
9. Teratogenicity	10	≥ 2
10. Mutagenicity	0	≥ 6
11. Carcinogenicity	10	≥ 2

(a): concern levels established for the listing of hazardous wastes.

(b): insufficient information on sublethal effects on plants.

APPENDIX D

ENVIRONMENTAL BEHAVIOUR AND TOXICITY OF ARSENIC

D.1 Chemical and physical properties of arsenic

Arsenic is a naturally occurring element in the soil. Pure arsenic is a grey-coloured metal. In the environment, arsenic is usually combined with either inorganic elements (i.e. oxygen, chlorine and sulphur) or organic elements (i.e. carbon and hydrogen). Many substances containing arsenic (both inorganic and organic) are naturally occurring, while others are manufactured. Since the inorganic forms of arsenic compounds are usually more toxic than the organic ones, it is important to mention the form of arsenic in toxicology (ATSDR, 1989).

In the wood preserving industry, the most common formulation is CCA (chromated copper arsenate) which is prepared from copper oxide, chromic and arsenic acids. Liquid concentrate (ranging from 50% to 70% concentration) is delivered in bulk (rail or tank truck) or in drums (125 kg) to the individual wood preserving plants. The concentration of active ingredients (for a stock solution of 50% concentrate) is 24% CrO_3 , 9% CuO and 17% As_2O_5 . The concentrate is freely soluble in water and is diluted to a working solution of 1% to 7% (Environment Canada, 1988). Arsenic can exist in the trivalent form (AsIII) or the pentavalent form (AsV). In CCA operations, arsenic is found in the pentavalent form (Environment Canada, 1988).

On contact of CCA preservative with wood, there is a sudden pH increase, ion-exchange and adsorption reactions of the CCA solution with the wood (Dahlgren and Hartford, 1972). The reactions are closely related to the presence of sapstain and glucose in the wood and the unfixed drippage from the treated lumber does not seem to be affected. In fact, the current practice is to recover and re-use solution from retorts and drippage areas.

Selected chemical and physical properties of elemental arsenic are summarized in Table D1.

D.2 Environmental behaviour of arsenic

D.2.1 Environmental mobility

The scoring criteria for environmental mobility are based on data from monitoring studies or on estimates of environmental partitioning derived from the Level 1 Fugacity Model (Mackay *et al.*, 1982). Fugacity models cannot handle inorganic chemicals (MOEE, 1991).

Consequently, assessment of environmental mobility of arsenic is based on available monitoring data.

Arsenic from anthropogenic sources is released into the environment mostly on land (81%), then in air (11%) and in surface water (5%). Arsenic emissions from natural sources represent about 10-15% of anthropogenic emissions. Volcano activities are by far the major source of natural arsenic emissions (ATSDR, 1989).

Arsenic in the environment may undergo a complex cycle of chemical conversions and transfers between media. Soluble forms of arsenic tend to be quite mobile in water, and less soluble species tend to adsorb to clay or soil particles. Microorganisms in soils, sediments and water can reduce and methylate arsenic to yield methyl arsines, which volatilize and re-enter the atmosphere (ASTDR, 1989).

Based on this information and the fact that inorganic ions are usually distributed among various media, environmental mobility for arsenic is scored at 10 (MOEE, 1991).

D.2.2 Persistence

Since arsenic is a natural element, it does not break down in the environment. Persistence of arsenic is scored at 10 (MOEE, 1991).

D.2.3 Bioaccumulation

Bioconcentration factors (BCF) have been measured for a few species. The reported BCF ranges from zero for rainbow trout to 17 for snail (*Helisoma campanulatum*) (MOEE, 1988; Spehar *et al.*, 1980).

Based on the BCF values, bioaccumulation is scored at 0 for arsenic.

D.2.4 Environmental exposure

Under present waste management practices (see Section 3.2 in Background Document), most of the process residuals are disposed of at a certified landfill. However, at some of the plants, drippage residuals are released into the environment by dripping on soil surface and by infiltration in groundwater.

The exposure pathway of concern is contamination of soil and groundwater. Mismanagement and improper disposal could result in substantial health and environmental adverse effects. Environmental exposure is scored at 10 for arsenic.

D.3 Toxicity of arsenic

The evaluation of toxicity of arsenic is based on the results of a literature search conducted in June 1988 by MOEE (1988) and the findings summarized in a toxicological profile prepared for ATSDR (1989).

D.3.1 Acute lethality

In animal studies, oral LD₅₀ values for various inorganic arsenic compounds have been reported to range from 10 to 300 mg/kg, with soluble compounds being more toxic than poorly soluble forms (NAS, 1977; USEPA, 1984). Harrison *et al.* (1958) reported the acute oral LD₅₀ for As₂O₃, given to different strains of mice by gavage, ranged from 15 to 39 mg/kg.

In aquatic toxicity, over 18 species of freshwater fish and aquatic invertebrates have been studied for inorganic arsenic. LC₅₀ for fish ranges from 12.6 mg/L for fathead minnow (*Pimephales promelas*) to 41.8 mg/L for bluegill (*Lepomis macrochirus*) (MOEE, 1988). Spehar *et al.* (1986) tested 30-day old fathead minnows in a flow-through test over a 96 hour period.

Based on this information, acute lethality for arsenic is scored at 6 (MOEE, 1991).

D.3.2 Sublethal effects, plants

Concentration of 2 mg/kg of arsenic in soil causes growth inhibition with continuous exposure. Concentration of 4 mg/kg or greater caused an irreversible reduction in cell proliferation (Pepper *et al.*, 1988). There is mention of the level of reduction in the study, although it is assumed to be more than 5%.

Effects of arsenic on aquatic plants are reported in terms of lethal effects only in the literature (MOEE, 1988). A 100% kill was reported on algae (*Cladophora sp.*) exposed to 2.3 mg/l over 2 weeks. A 95% kill was observed on *Potamogeton sp.* exposed to 2.3 mg/l over one month (Cowell, 1965).

Based on this limited information, sublethal effects on plants is scored at 6, based on Pepper *et al.* (1988) findings.

D.3.3 Sublethal effects, mammals

Long-term oral exposure may lead to anaemia, peripheral neuropathy, cardiotoxicity, and a group of skin disorders characterized by hyperpigmentation and hyperkeratosis.

Mizuta *et al.*, (1956) reported anaemia and leucopenia in adults ingesting about 3 mg/day in contaminated soy sauce. At a standard weight of 70 kg for an adult, the dose is equivalent to 0.04 mg/kg.

Arsenic-induced peripheral neuropathy can sometimes be detected by electromyographic (EMG) techniques. Characteristic changes include decreased nerve conduction amplitude with little change in nerve conduction velocity (NCV) (Donofrio and Wilborn, 1985). Hindmarsh *et al.*, (1977), studying a group of patients exposed to elevated levels of inorganic arsenic in drinking water (above 0.1 mg/l), observed EMG abnormalities in half the cases. Valentine *et al.*, (1981) measured NCVs in residents of several western communities where drinking water levels of arsenic ranged from 0.05 to 0.387 mg/l. Although most findings are in the normal range, a significant decrease in the ulnar sensory nerve function was noted in both males and females. Similar results were reported by Blom *et al.*, (1985), who concluded that workplace exposure to air levels below 0.05 mg/m³ did not result in any clinically significant neuropathy.

Chronic inhalation exposure results in irritation to the mucous membranes of the eyes and nasopharynx (Vallee *et al.*, 1960). A hoarse voice is a common sign in arsenic workers, and a perforated nasal septum may occur following prolonged exposure. No such effects, however were noted in workers exposed to 0.2 mg/m³ in air (ACGIH, 1986).

Sublethal effects on mammals is scored at 10.

D.3.4 Sublethal effects, non-mammals

Maximum acceptable toxicant concentrations (MATC) have been determined experimentally for cladoceran (*Daphnia magna*) and 3 species of fish (MOEE, 1988). MATC levels range from 0.91 mg/l for cladoceran (Lima *et al.*, 1984), 1.14 mg/l for rainbow trout (*S. gairdneri*) (Mayer *et al.*, 1986), to 3.03 mg/l for fathead minnow (*Pimephale promelas*) (Call

et al., 1983)

Base of MATC levels, sublethal effects on non-mammals is scored at 2 for arsenic (MOEE, 1991).

D.3.5 Teratogenicity

Intraperitoneal exposure of 10 to 45 mg/kg/day of sodium arsenate to rats, mice or hamsters during gestation has been reported to increase the frequency of a number of fetal malfunctions (ATSDR, 1989; Ferm and Carpenter, 1968; Ferm *et al.*, 1971; Hood and Bishop, 1972; Burk and Beaudoin, 1977; Willhite, 1981; Ferm and Hanlon, 1985). By the oral route, arsenate is much less toxic. Dose of 120 mg/kg administered by gavage to mice during gestation caused decreased birth weight, and increased prenatal mortality, but no fetotoxic or teratogenic effects were seen with oral doses up to 100 mg/kg (Hood *et al.*, 1977; 1978).

Similar results have been reported for sodium arsenite, although the teratogenic and fetotoxic potential of arsenite appears to be somewhat greater than for arsenate. Baxly *et al.*, (1981) administered single oral doses of sodium arsenite to pregnant mice and observed no discernable teratogenic or maternal toxicity at doses of 20 mg/kg. Doses of 40 to 45 mg/kg resulted in both maternal lethality and fetotoxicity (decreased fetal weight and increased fetal resorption), with a low incidence of gross malformations. Similar findings were reported by Hood *et al.* (1982) who administered single oral doses of sodium arsenite by gavage to pregnant hamsters. Doses of 20 mg/kg given on days 9, 10, or 11 produced no significant fetotoxicity, while doses of 25 mg/kg given on day 8 or day 12 lead to increased parental mortality and decreased fetal weight.

Based on this information, teratogenicity is scored at 4.

D.3.6 Mutagenicity

Arsenic compounds have been found to be inactive or very weak in producing gene mutations. However, trivalent and pentavalent arsenic are capable, at least *in vitro*, of causing chromosomes breaking. *In vivo* studies have yielded conflicting results for chromosomal damage. Assays for DNA repair/damage also have yielded mixed results (MOEE, 1988).

Both arsenate and arsenite have been found to be related to chromosome aberration and sister chromatid exchange (SCE) in cultured animal and human cells tested *in vitro* (Jacobson-Kram and Montalbano, 1985). Larramendy *et al.* (1981) incubated Syrian hamsters embryo cells and human peripheral lymphocytes with low concentrations (near 10^{-5} M) of arsenate and

arsenite, and observed significant increases in chromosome aberration frequency with both compounds and in both cell types. Arsenite was about ten times more effective than arsenate. Dose-related increases in SCE were also reported. Similar results have been reported by Nakamuro and Sayato (1981), Nordenson *et al.* (1981), Wan *et al.* (1982) and Lee *et al.* (1985).

Mutagenicity is scored at 8, based on the information reported above.

D.3.7 Carcinogenicity

Many studies have examined the effects of arsenic exposure on humans (MOEE, 1988). Exposures by contaminated drinking water or occupation have been examined (USEPA, 1984). Exposures by oral route have been found to induce changes in skin epithelium which develop from hyperpigmentation to hyperkeratosis or squamous cell carcinomas (Tseng, 1977; Borgono *et al.*, 1977). Occupational exposure to arsenic dusts, during smelting or other mineral/metal processing, have been found to lead to respiratory tract cancers (Brown *et al.*, 1983; Lee-Feldstein, 1986).

In animals, carcinogenicity has not been observed following oral or inhalation exposures (MOEE, 1988).

Under the IARC classification, arsenic and certain arsenic compounds are classified in Group 1 (IARC, 1982).

Based on the information above and IARC classification, arsenic is scored at 10.

D.4 Results of Vector Scoring System of arsenic

The results of the vector scoring process are presented in Table D2. The results indicate that arsenic scoring is above the concern level in all of the parameters examined, except for bioaccumulation and sublethal effects in non-mammals.

D.5 References

ACGIH, 1986.

Committee on the threshold limit values: documentation of threshold limit values and biological exposure indices, ed. 5, Cincinnati, OH. American Conference of Governmental Industrial Hygienists (ACGIH).

ATSDR, 1989.

Toxicological profile for arsenic. Prepared by Life Systems Inc. for the Agency for Toxic Substances and Disease Registry (ATSDR), U.S. Public Health Service, in collaboration with USEPA. March, 1989. ATSDR/TP-88/02. PB89-185706.

Baxley, M.N., R.D. Hood, G.C. Vedel, W.P. Harrison and G.M. Szczech, 1981.

Prenatal toxicity of orally administered sodium arsenite in mice. *Bull. Environ. Contam. Toxicol.* 26:749-756.

Blom, S., B. Lagerkvist and H. Linderholm, 1985.

Arsenic exposure to smelter workers: clinical and neurophysiological studies. *Scand. J. Work Environ. Health*, 11:265-269.

Borgono, P.M., *et al.*, 1977.

Arsenic in the drinking water of the City of Antofagasta: epidemiological and clinical study before and after the installation of the treatment plant. *Environ. Health Perspectives*, 19:103-105.

Brown, C.C. and K.C. Chu, 1983.

A new method for the analysis of cohort studies: implications of the multistage theory of carcinogenesis applied to occupational arsenic exposure. *Environ. Health Perspectives*, 50:293-308.

Burk, D., and A.R. Beaudoin, 1977.

Arsenate-induced renal agenesis in rats. *Teratology*. 16:247-260.

Call, D.J., L.T. Brooke, N. Ahmad and J.E. Richter, 1983.

Toxicity and metabolism studies with EPA priority pollutants and related chemicals in freshwater organisms. PB83-263665. National Technical Information Service, Springfield, Virginia.

Cowell, B.C., 1965.

The effects of sodium arsenite and silvex on the plankton populations in farm ponds. *Trans. Am. Fish. Soc.* 94. 371.

- Dahlgren, S.E. and W.H. Hartford, 1972.
Kinetics and mechanism of fixation of Cu-Cr-As wood preservatives. *Holzforschung*, 26(2), 62-69; 26(3), 105-113; 26(4), 142-149.
- Donofrio, P.D. and A.J. Wilborn, 1985.
Subacute arsenic toxicity presenting on clinical and electromyographic examination as Guillain-Barre syndrome. 110th Annual Meeting of the American Neurological Association, Chicago, IL, October 2-5, 1985. *Ann. Neurol.* 18:156-157.
- Environment Canada, 1988.
Chromated copper arsenate wood preservation facilities: recommendations for design and operation. Prepared by Envirochem Services for Conservation and Protection, Environment Canada. Report EPS 2/WP/3, April 1988.
- Ferm, V.H. and S.J. Carpenter, 1968.
Malformations induced by sodium arsenate. *J. Reprod. Fert.* 17:199-201.
- Ferm, V.H., A. Saxon and B.M. Smith, 1971.
The teratogenic profile of sodium arsenate in the golden hamster. *Arch. Environ. Health.* 22:557-560.
- Ferm, V.H., and D.P. Hanlon, 1985.
Constant rate exposure of pregnant hamsters to arsenate during early gestation. *Environ. Res.* 37: 425-432.
- Harrison, J.W., E.W. Packman and D.D. Abbott, 1958.
Acute oral toxicity and chemical and physical properties of arsenic trioxides. *Arch. Ind. Health*, 17:118-123
- Hindmarsh, J.T., O.R. McLetchie, L.P. Heffernan, O.A. Haynie, H.A. Ellenberger, R.F. McCurdy and H.J. Thiebaut, 1977.
Electromyographic abnormalities in chronic environmental arsenicalism. *J. Anal. Toxicol.* 1:270-276.
- Hood, R.D. and S.L. Bishop, 1972.
Teratogenic effects of sodium arsenate in mice. *Arch Environ Health.* 24:62-65.
- Hood, R.D., G.T. Thacher and B.L. Patterson, 1977.
Effects in the mouse and rat of prenatal exposure to arsenic. *Environ. Health Perspect.* 19:219-222.
- Hood, R.D., G.T. Thacher, B.L. Patterson and G.M. Szczech, 1978.
Prenatal effects of oral versus intraperitoneal sodium arsenate in mice. *J Environ. Pathol. Toxicol.* 1:857-864.

- Hood, R.D., W.P. Harrison and G.C. Vedel, 1982.
Evaluation of arsenic metabolites for prenatal effects in the hamster. *Bull. Environ. Contam. Toxicol.* 29:679-687.
- IARC, 1982.
IARC monograph on the evaluation of the carcinogenic risk of chemicals to humans. Supplement 29, International Agency for Research on Cancer, Lyon, France.
- Jacobson-Kram, D. and D. Montalbano, 1985.
The reproductive effects assessment groups's report on the mutagenicity of inorganic arsenic. *Environ. Mutagen.* 77:787-804.
- Larramendy, M.L., N.C. Popescu and J. DiPaolo, 1981.
Induction by inorganic metal salts of sister chromatid exchanges and chromosome aberrations in human and Syrian hamster strains. *Environ. Mutagen.* 3:597-606.
- Lee, T.C., M. Oshimura and J.C. Barrett, 1985.
Comparison of arsenic-induced transformation, cytotoxicity, mutation and cytogenetic effects in Syrian hamster embryo cells in culture. *Carcinogenesis (Lond.)*, 6:14221-1426.
- Lee-Feldstein, A., 1986.
Cumulative exposure to arsenic and its relationship to cancer among copper smelter employees. *J. Occup. Med.* 28: 296-302.
- Lima, A.R., C. Curtis, D.E. Hammermeister, T.P. Markee, C.E. Northcott and L.T. Brooke, 1984.
Acute and chronic toxicities of arsenic(III) to fathead minnows, flatfish, daphnids, and an amphipod. *Arch. Environ. Contam. Toxicol.* 13: 595-601.
- Mackay, D. and S. Paterson, 1982.
Fugacity revisited. *Env. Sc. and Tech.*, Vol.16, No.12, 1982.
- Mayer, F.L., K.S. Mayer and M.R. Ellersieck, 1986.
Relation of survival to other endpoints in chronic toxicity tests with fish. *Env. Toxicol. Chem.* 5: 737-748.
- Mizuta, N., M. Mizuta, F. Ita, T. Ito, H. Uchida, Y. Watanabe, H. Akama, T. Murakami, F. Hayashi, K. Nakamura, T. Tamaguchi, W. Mizuiz, S. Oishi and H. Matsumura, 1956.
An outbreak of acute arsenic poisoning caused by arsenic-contaminated soy sauce (shoye). A clinical report of 220 cases. *Bull Yamaguchi Med. Sch.* 4:131-150.

MOEE, 1988.

CESARS (Chemical Evaluation Search and Retrieval System) dossier on arsenic. Prepared by Ontario Ministry of Environment and Energy, provided by Canadian Centre for Occupational Health and Safety. June 1988.

MOEE, 1991.

Guidance manual for hazardous waste categorization and review program. Prepared by Waste Management Branch, Ontario Ministry of Environment and Energy (MOEE), January 1991.

Nakamura, M. and Y. Sayato, 1981.

Comparative studies of chromosomal aberrations induced by trivalent and pentavalent arsenic. *Mutat. Res.* 88:73-80.

NAS (National Academy of Science), 1977.

Arsenic. Prepared by the Committee on Medical and Biological Effects of Environmental Pollutants, Division of Medical Sciences, National Research Council. Washington DC.

Nordenson, I., A. Sveins and L. Beckman, 1981.

Chromosome aberrations in cultured human lymphocytes exposed to trivalent and pentavalent arsenic. *Scand. J. Work Environ. Health*, 7:277-281.

Pepper, I., N. Galanti and J. Sans, 1988.

Reversible inhibition of root growth and cell proliferation by pentavalent arsenic in *Allium cepa* L. *Environ. Exp. Bot.* 28: 9-18.

Spehar, R.L., J.T. Fiandt, R.L. Anderson and D.L. DeFoe, 1980.

Comparative toxicity of arsenic compounds and their accumulation in invertebrates and fish. *Arch. Environ. Contam. Toxicol.* 9: 53-63.

Spehar, R.L. and J.T. Fiandt, 1986.

Acute and chronic effects of water quality criteria-based metals mixtures on three aquatic species. *Env. Toxicol. Chem.* 5: 917-931.

Tseng, W.P., 1977.

Effects and dose-response relationships of skin cancer and Blackfoot disease with arsenic. *Environ. Health Perspect.* 19: 109-119.

USEPA, 1984.

Health assessment document for inorganic arsenic. Final Report. Research Triangle Park, N.C.: United States Environmental Protection Agency. EPA-600/8-83-021F.

- Valentine, J.L., D.S. Campion, M.D. Schluchter and F.J. Massey, 1981.
Arsenic effects on human nerve conduction. IN: Howell, J.C., J.M. Hawthorne and L. White (ed). Trace element metabolism in man and animals. Canberra: Australian Academy of Science, pp 409-411.
- Vallee, B.L., D.D. Ulmer and W.E.C. Wacker, 1960.
Arsenic toxicology and biochemistry. Arch. Ind. Health. 21:132-151.
- Wan, B., R.T. Christian and S.W. Soukup, 1982.
Studies of cytogenetic effects of sodium arsenicals on mammalian cells *in vitro*. Environ. Mutag. 4:493-498.
- Willhite, C.C., 1981.
Arsenic-induced axial skeletal (dysraphic) disorders. Exp. Mol. Pathol. 34: 145-158.

Table D1: Properties of arsenic

Chemical Abstract #	: 7440-38-2
Chemical Abstract Name	: arsenic
Synonyms	: arsenic-75 metallic-arsenic arsenic black colloidal arsenic
Chemical Formula	: As
Molecular Weight	: 74.92
Boiling Point	: 613°C (sublimation)
Melting Point	: 817°C (at 28 atmospheres)
Density	: 5.727 g/cm ³
Solubility (1)	: insoluble

(1) Solubility of various salts:

arsenic pentoxide:	freely soluble
arsenic trioxide:	2.1 mg/L
calcium arsenate:	0.013 mg/L
sodium arsenate:	very soluble
sodium arsenite:	freely soluble

Table D2: Results of Vector Scoring of arsenic

<u>Parameter</u>	<u>Score</u>	<u>Concern Levels (a)</u>
1. Environmental Mobility	10	≥ 7
2. Environmental Persistence	10	≥ 7
3. Bioaccumulation	0	≥ 7
4. Environmental Exposure	10	≥ 7
5. Acute lethality	8	≥ 6
6. Sublethal effects plants	6	≥ 4
7. Sublethal effects mammals	10	≥ 4
8. Sublethal effects non-mammals	2	≥ 4
9. Teratogenicity	4	≥ 2
10. Mutagenicity	8	≥ 6
11. Carcinogenicity	10	≥ 2

(a): concern levels established for the listing of hazardous wastes.

APPENDIX E

ENVIRONMENTAL BEHAVIOUR AND TOXICITY OF CHROMIUM

E.1 Chemical and physical properties of chromium

Selected physical and chemical properties of elemental chromium are listed in Table E1.

Chromium is a metallic element which does not occur naturally in its elemental form.

However, chromium is found in nature primarily as a mineral chromite. Chromium exists in four oxidation states. But only two of them, Cr(III) and Cr(VI), are considered important, due to their predominance and stability in the ambient environment.

Trivalent chromium Cr(III) is the most stable oxidation state, and the most important chemically. It has a strong tendency to form very stable complexes with negatively charged organic or inorganic species. Hence, it is unlikely that appreciable quantities of uncomplexed Cr^{+3} will be found in aqueous solution as long as any anionic dissolved or particulate matter (e.g. decaying plant or animal tissue, silt or clay particles) is suspended therein. Even if there are no anionic species present, Cr can react with water itself in neutral solution to form colloidal hydrous oxides. Below pH 5, the Cr^{+3} hexaquo complex is stable. Above pH 9, soluble hydroxides are formed (NRC, 1976).

Hexavalent chromium Cr(VI) in aqueous solution exists almost exclusively in the form of oxo anions (CrO_4^{-2} , $\text{Cr}_2\text{O}_7^{-2}$). Its chemistry is different from that of trivalent chromium (Cr^{+3}). In dilute solution (< 1 ppm), the predominant form is CrO_4^{-2} which, being negatively charged, does not complex with anionic particulate matter. Hence, Cr(VI) is more mobile than Cr(III). Cr(VI) is largely associated with particulate matter and it is subject to sedimentation and filtration. However Cr(VI) compounds are more powerful oxidizing agents, especially in acidic solutions. The tendency is strong to react with oxidizable matter to form Cr(III). Nevertheless, if the concentration of oxidizable substances is low in the water, then Cr(VI) may persist for considerable periods of time (NRC, 1976).

In the wood preserving industry, the most common formulation is CCA (chromated copper arsenate) which is prepared from copper oxide, chromic and arsenic acids. Liquid concentrate (ranging from 50% to 70% concentration) is delivered in bulk (rail or tank truck) or in drums (125 kg) to the individual wood preserving plants. The concentration of active ingredients (for a stock solution of 50% concentrate) is 24% CrO_3 , 9% CuO and 17% As_2O_5 . The concentrate is freely soluble in water and is diluted to a working solution of 1% to 7% (Environment Canada, 1988). The chromium in the chromated copper arsenate (CCA) solution is found in the hexavalent form of Cr(VI) (Environment Canada, 1988; NRC, 1976).

On contact of CCA preservative with wood, there is a sudden pH increase, ion-exchange and adsorption reactions of the CCA solution with the wood (Dahlgren and Hartford, 1972). The reactions are closely related to the presence of sapstain and glucose in the wood and the unfixed drippage from the treated lumber does not seem to be affected. In fact, the current practice is to recover and re-use solution from retorts and drippage areas.

Chromium in ambient air is normally associated with airborne soil particles and industrial air emissions.

E.2 Environmental behaviour of chromium

E.2.1 Environmental mobility

Chromium released to the environment from combustion processes and metallurgical industries is present mainly as Cr_2O_3 . Chromium released to the environment from chromate manufacturing and applications largely contains bio-available Cr(VI). Chromium is primarily removed from the atmosphere by fallout and precipitation. The residence time of chromium in the atmosphere is expected to be less than 10 days (Nriagu, 1979).

Because there are no known chromium compounds that can volatilize from water, transport of chromium from water to the atmosphere is not likely, other than by transport by wind blown sea spray. Most of the Cr(III) is eventually expected to precipitate in sediments. Small amounts of Cr(III) may remain in solution as soluble complexes. Cr(VI) will predominantly be present in soluble form. These soluble forms of chromium may be stable enough to undergo intra-media transport. However, Cr(VI) will eventually be reduced to Cr(III) by organic matters present in water (ATSDR, 1989, 1991).

Since the Fugacity Model (Level 1) cannot be used to assess the relative mobility of chromium in different media (fugacity modelling is not suitable for inorganic chemicals), the scoring is based on monitoring data. Data on environmental fate of chromium indicate that near 75% of total Cr(VI) particles have diameters of less than 10 micrometers. This suggests that the particles can be dispersed in several media (from air to soil and surface water). Based on this assessment, chromium is scored at 10 for environmental mobility (MOEE, 1991a, 1991b).

E.2.2 Persistence

Persistence is best evaluated in terms of half-life of the contaminant in a specific medium, under known conditions. This type of information is limited in the case of chromium.

In water, chromium (VI) is eventually reduced to chromium (III) by organic matter. The residence time of total chromium in lake water ranges from 4.6 to 18 years (Fishbein, 1981; Schmidt and Andren, 1984).

In soil, chromium may be transported to the atmosphere in the form of aerosol. Runoff and leaching may transport chromium from soil to surface waters and groundwaters. Flooding of soils and the subsequent anaerobic decomposition of plant matters may increase mobilization of chromium in soils due to formation of soluble complexes. The half-life of chromium in soils may be several years (USEPA, 1984; 1985).

Although there is limited adequate information for this parameter, persistence is scored at 10 for chromium (MOEE, 1991a).

E.2.3 Bio-accumulation

One of the parameters frequently used to express bio-accumulation is the bio-concentration factor (BCF). The bioconcentration factor (BCF) for Cr(VI) in rainbow trout (*Salmo gairdneri*) is approximately 1. In bottom-feeder species, such as the oyster (*Crassostrea virginica*), blue mussel (*Mytilus edulis*), and soft shell clam (*Mya arenaria*), the BCF values for Cr(III) and Cr(VI) may range from 86 to 192 (Fishbein, 1981; Schmidt and Andren, 1984).

The score of 4 is based on a BCF of 200 reported for an unspecified freshwater fish. It is uncertain whether the BCF is representative of the bio-concentration potential of chromium, considering that significantly higher values are reported in numerous other studies (MOEE, 1991a).

E.2.4 Environmental exposure

Under present waste management practices (see Section 3.2 in Background Document), most of the process residuals are disposed of at a certified landfill. However, at some of the plants, drippage residuals are released into the environment by dripping on soil surface and infiltration in groundwater.

The exposure pathway of concern is contamination of groundwater. Mismanagement and improper disposal could result in substantial health and environmental adverse effects. Environmental exposure is scored at 10 for chromium.

E.3 Toxicity of chromium

The main source of information for evaluating the toxicity of chromium is derived from a preliminary assessment completed in August 1991 by the Hazardous Contaminants Branch (MOEE, 1991a). The document include the results of literature search (AQUIRE, 1991) conducted in January 1991, the results of toxicological profile on chromium from the U.S. Department of Health and Human Services (ATSDR, 1991), and the results of a synoptic review from the U.S. Fish and Wildlife Service (Eisler, 1986).

The toxicity of Cr(III) and the one of Cr(VI) differ considerably. Whenever reported in the toxicity studies, the form of Cr is reported in the findings. The vector scoring always looks at the most toxic form of Cr, which is Cr(VI) in the majority of the toxic effects.

E.3.1 Acute lethality

A vector score of 8 is based on a 96-hour LC50 aquatic exposure of 0.650 mg/L chromium (VI) reported for bryozoa (*plumatella sp.*) (Pardue, 1980). Numerous other studies using oral, inhalation and dermal exposure of a variety of soluble chromium compounds suggest lower scores: scores of 6 for oral and inhalation and 4 for dermal exposure (MOEE, 1991a; AQUIRE, 1991; Eisler, 1986).

E.3.2 Sublethal effects, plants

A score of 8 is based on several studies reporting a 35% decrease in population growth in several species of dinoflagellates, diatoms and haptophytes (MOEE, 1991a). Reported concentrations were as low as 0.01 mg/L (Wilson, 1980).

E.3.3 Sublethal effects, mammals

A score of 10 is based on inhalation NOAELs (no observable adverse effect level) reported for humans (nasal effects) at 0.001 mg/m³ chromium (VI) (Lindberg and Hedenstierna, 1983). Glaser *et al.* (1986, 1988) also reported inhalation NOAELs for rat (immune system) at 0.1 mg/m³ chromium (VI). Oral studies report NOAELs which support a lower score of 6 (MOEE, 1991a; ATSDR, 1991).

E.3.4 Sublethal effects, non-mammals

A score of 4 is based on several studies reporting MATCs (maximum acceptable toxicant concentration) in freshwater species. MATCs as low as 0.051 mg/L, and ranging to 0.105 mg/L, were reported for rainbow trout (*Salvelinus fontinalis*) exposed to chromium (VI) in water with hardness of 34 mg CaCO₃/L (Sauter *et al.*, 1976). MATCs ranging from 0.047 mg/L to 0.093 mg/L were reported for water flea (*Daphnia magna*) exposed to chromium (III) in freshwater (USEPA, 1980).

E.3.5 Teratogenicity

A score of 8 is based on the incidence of terata in chickens. Embryolethal and teratogenic effects have been observed in the range of 0.2 mg/kg (Gilani and Marano, 1979) to 1.7-22.9 mg/kg (Ridgeway and Karnofsky, 1952). The findings are however questionable, because the routes of exposure are not well defined, the daily dosages not identified, and the time of gestation is not indicated (MOEE, 1991a).

E.3.6 Mutagenicity

A score of 10 is based on evidence of mutations and interaction with genetic material in mammalian and non-mammalian *in vivo* test systems, and evidence of mutagenic responses *in vitro* (MOEE, 1991a). The exposure route was by inhalation only and for Cr(VI). Positive evidence of mutagenicity was observed in hamster (Sugiyama *et al.*, 1986; Levis and Majone, 1979; MacRae *et al.*, 1979; Montaldi *et al.*, 1987; Venier *et al.*, 1982; Newbold *et al.*, 1979), mouse (Raffetto *et al.*, 1977; Fornace *et al.*, 1981; Shindo *et al.*, 1989), and drosophila systems (*drosophila melangaster*) *in vivo* (Gava *et al.*, 1989; Rasmuson, 1985; Rodriquez-Arnaiz and Martinez, 1986; Zimmering *et al.*, 1985), and in human *in vitro* (Fornace *et al.*, 1981; Douglas *et al.*, 1980; Gomez-Arroyo *et al.*, 1981; Montaldi *et al.*, 1987; Sarto *et al.*, 1980; Stella *et al.*, 1982).

E.3.7 Carcinogenicity

The score of 10 is based on several studies reporting increased incidence of tumors of the respiratory system in mice, rats, and humans following prolonged exposure (by inhalation only) to chromium and chromium compounds. Tumorigenesis was primarily attributed to Cr(VI) (MOEE, 1991a).

In the chromate producing industry, workers exposed to 0.03-1.1 mg/m³ Cr over 4 to 24 years have developed respiratory cancer. In the chromate pigment production industry, workers exposed to an estimated Cr(VI) concentration of 0.5-1.5 mg/m³ for 6 to 9 years have developed respiratory cancer (Post and Campbell, 1980).

Chromium has been classified a Class A human carcinogen by USEPA (IRIS, 1990).

E.3.8 Results of vector scoring of chromium

The results of the vector scoring for chromium are summarized in Table E2.

All parameters associated with the environmental behaviours of chromium are scored high, except for bioaccumulation. Combined with toxicity parameters all scored above concern levels, chromium is considered a high risk for human health and the environment.

E.4 References

AQUIRE, 1991.

Aquatic Information Retrieval Computerized Database. Computerized database developed by USEPA. Search done in January 1991.

ATSDR, 1989.

Toxicological profile for chromium. Prepared by Syracuse Research Corporation for the Agency for Toxic Substances and Disease Registry (ATSDR), U.S. Department of Public Health and Human Service. July 1989. PB89-236665.

ATSDR, 1991.

Draft (update) toxicological profile for chromium. Prepared by Syracuse Research Corporation for the Agency for Toxic Substances and Disease Registry (ATSDR), U.S. Department of Public Health and Human Service, October 1991.

Dahlgren, S.E. and W.H. Hartford, 1972.

Kinetics and mechanism of fixation of Cu-Cr-As wood preservatives. *Holzforschung*, 26(2), 62-69; 26(3), 105-113; 26(4), 142-149.

Douglas, G.R., R.D.L. Bell and C.E. Grant, 1980.

Effect of lead chromate on chromosome aberration, sister-chromatid exchange and DNA damage in mammalian cells *in vitro*. *Mutat. Res.* 77:157-163.

Eisler, R., 1986.

Chromium hazards to fish, wildlife, and invertebrates: a synoptic review. Fish and Wildlife Service, U.S. Department of the Interior, Biological report 85(1.6), January 1986.

Environment Canada, 1988.

Chromated copper arsenate wood preservation facilities: recommendations for design and operation. Prepared by Envirochem Services for Conservation and Protection, Environment Canada. Report EPS 2/WP/3, April 1988.

Fishbein, L., 1981.

Sources, transport and alterations of metal compounds: an overview. I: Arsenic, beryllium, cadmium, chromium and nickel. *Environ Health Perspect* 40:43-64.

Fornace, A. J. Jr., D.S. Seres and J.F. Lechner, 1981.

DNA-protein cross-linking by chromium salts. *Chem. Biol. Interact.* 36:345-354.

- Gava, C., R. Costa and M. Zordan, 1989.
Induction of gene mutations in *Salmonella* and *Drosophila* by soluble Cr(VI) compounds: synergistic effects of nitrilotriacetic acid. *Toxicol. Environ. Chem.* 22:27-38.
- Gilani, S.H. and M. Marano, 1979.
Chromium poisoning and chick embryogenesis. *Environ. Res.* 19:427-431.
- Glaser, U., D. Hochrainer and H. Kloppel, 1986.
Carcinogenicity of sodium dichromate and chromium (VI/III) oxide aerosols inhaled by male Wistar rats. *Toxicology* 42:219-232.
- Glaser, U., D. Hochrainer and H. Oldiges, 1988.
Investigations of the lung carcinogenic potentials of sodium dichromate and Cr(VI/III) oxide aerosols in Wistar rats. *Environ. Hyg.* 1:111-116.
- Gomez-Arroyo, S., M. Altamirano and R. Villalobos-Pietrini, 1981.
Sister-chromatid exchanges induced by some chromium compounds in human lymphocytes *in vitro*. *Mutat. Res.* 90:425-431.
- IRIS, 1990.
Integrated Risk Information System (computerized, peer-reviewed database of USEPA), search done February 1990.
- Levis, A.G. and F. Majone, 1979.
Cytotoxic and clastogenic effects of soluble chromium compounds on mammalian cell cultures. *Br. J. Cancer* 40:523-533.
- Lindberg, E. and G. Hedenstierna, 1983.
Chrome plating: symptoms, findings in the upper airways, and effects on lung function. *Arch. Environ. Health* 9:333-340.
- MacRae, W.D., R.F. Whiting and H.F. Stich, 1979.
Sister chromatid exchanges induced in cultured mammalian cells by chromate. *Chem. Biol. Interact.* 26:281-286.
- MOEE, 1991a.
Chromium: Ontario environmental assessment; preliminary assessment. Search done January 1991, for Hazardous Contaminants Branch, Ontario Ministry of Environment and Energy.

MOEE, 1991b.

Guidance manual for hazardous waste categorization and review program. Waste Management Branch, Ontario Ministry of Environment and Energy. January 1991.

Montaldi, A., L. Zentilin and M. Zordan, 1987.

Chromosomal effects of heavy metals (Cd, Cr, Hg, Ni and Pb) on cultured mammalian cells in the presence of nitrilotriacetic acid (NTA). *Toxi. and Envi. Chem.* 14:183-200.

Newbold, R.F., J. Amos and J.R. Connell, 1979.

The cytotoxic, mutagenic and clastogenic effects of chromium-containing compounds on mammalian cells in culture. *Mutat. Res.* 67:55-63.

NRC, 1976.

Effect of Chromium in The Canadian Environment, Associate Committee on Scientific Criteria for Environmental Quality, ISSN 0316 - 0114.

Nriagu, J.A., 1979.

Copper in the atmosphere and precipitation. In: Nriagu J.A., ed. Copper environment. New York, NY: John Wiley and Sons, 43-75.

Pardue, W.J. and T.S. Wood, 1980.

Baseline toxicity data for freshwater bryozoa exposed to copper, cadmium, chromium and zinc. *J. Tenn. Acad. Sci.* 55(1):27-31.

Post, M.A. and P.G. Campbell, 1980.

Lead chromate pigments - a literature survey on environmental and toxic effects. U.S. Dep. Comm. Nat. Bur. Stand. Rep. NBSIR 80-1974. 38p.

Raffetto, G., S. Parodi and C. Parodi, 1977.

Direct interaction with cellular targets as the mechanism for chromium carcinogenesis. *Tumori.* 63:503-512.

Rasmuson, A., 1985.

Mutagenic effects of some water-soluble metal compounds in a somatic eye-color test system in *Drosophila melanogaster*. *Mutat. Res.* 157:157-162.

Ridgeway, L.P. and D.A. Karnofsky, 1952.

The effects of metals on the chick embryo: toxicity and production of abnormalities in development. *Ann. N.Y. Acad. Sci.* 55:203-215.

Rodriguez-Arnaiz, R. and R.F.M. Martinez, 1986.

Genetic effects of potassium dichromate and chromium trioxide in *Drosophila melanogaster*. *Cytologia* 51:421-425.

- Sarto, F., A.G. Levis and C. Paulon, 1980.
Clastogenic activity of hexavalent and trivalent chromium in cultured human lymphocytes. *Caryologia* 33:239-250.
- Sauter, S., K.S. Buxton, K.J. Macek and S.R. Petrocelli, 1976.
Effects of exposure to heavy metals on selected freshwater fish. Toxicity of copper, cadmium, chromium and lead to eggs and fry of seven fish species. USEPA report 600/3-76-105. 75p.
- Schmidt, J.A. and A.W. Andren, 1984.
Deposition of airborne metals into the Great Lakes: an evaluation of past and present estimates. *Advances in Environmental Science Technology* 14:81-103.
- Shindo, Y., Y. Toyoda and K. Kawamura, 1989.
Micronucleus test with potassium chromate (VI) administered intraperitoneally and orally to mice. *Mutat. Res.* 223:403-406.
- Stella, M., A. Montaldi and R. Rossi, 1982.
Clastogenic effects of chromium on human lymphocytes *in vitro* and *in vivo*. *Mutat. Res.* 101:151-164.
- Sugiyama, M., S.R. Patierno, O. Cantoni, 1986.
Characterization of DNA lesions induced by CaCrO_4 in synchronous and asynchronous cultured mammalian cells. *Mol. Pharmacol.* 29:606-613.
- USEPA, 1980.
Ambient water quality criteria for chromium. USEPA report 440/5-80-035.
- USEPA, 1985.
Environmental profiles and hazard indices for constituents of municipal sludge: chromium. Office of Water Regulations and Standards, EPA, Washington, DC.
- Venier, P., A. Montaldi and F. Majone, 1982.
Cytotoxic, mutagenic and clastogenic effects of industrial chromium compounds. *Carcinogenesis* 3(11):1331-1338.
- Wilson, W.B. and L.R. Freeburg, 1980.
Toxicity of metals to marine phytoplankton cultures. *Ecol. Res. Ser.* (EPA-600/3-80-025), USEPA, Environ. Res. Lab., Narragansett, RI. (NTIS PB80-182843:110p).
- Zimmering, S., J.M. Mason and R. Valencia, 1985.
Chemical mutagenesis testing in *Drosophila*. II. Results of 20 coded compounds tested for the National Toxicology Program. *Environ. Mutagen.* 7:87-100.

Table E1: Properties of elemental chromium

Chemical Abstract #	: 7440-47-3
Chemical Abstract Name	: Chromium
Chemical Formula	: Cr
Molecular Weight	: 51.996
Boiling Point	: 2,672°C
Melting Point	: 1,857°C
Density	: 7.20 g/cm ³ (28°C)
Solubility	: insoluble
Volatility	: 1 mm Hg at 1616°C
Flash Point (open cup)	: unknown
Conversion Factor	: 1 ppm = 1 mg/L

Table E2: Results of Vector Scoring of chromium

<u>Parameter</u>	<u>Score (b)</u>	<u>Concern Levels (a)</u>
1. Environmental Mobility	10	≥ 7
2. Environmental Persistence	10	≥ 7
3. Bioaccumulation	4	≥ 7
4. Environmental Exposure	10	≥ 7
5. Acute lethality	8	≥ 6
6. Sublethal effects plants	8	≥ 4
7. Sublethal effects mammals	10	≥ 4
8. Sublethal effects non-mammals	4	≥ 4
9. Teratogenicity	8	≥ 2
10. Mutagenicity	10	≥ 6
11. Carcinogenicity	10	≥ 2

(a): concern levels established for the listing of hazardous wastes.

(b): the score is based on the most toxic form of Cr which is hexavalent chromium (CrVI).

TP
996
.W6
C38
1994

Categorization (listing)
background document : wood
preservation wastes,

77264